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Original Articles.

THE CEREBRAL BLOOD-VESSELS IN HEALTH AND DISEASE.

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THE great progress which microscopical science has made in the last ten years,—in which I include the method of hardening, preserving, staining, &c., as also the improvement in the optical part of instruments,—has taught us not only to know more about the finer structures of animal tissues and their elements, but has also revealed an increasing number of changes, which denote the impress of a pathological condition.

Latterly certain sceptical and unprejudiced investigators have undertaken to verify more exactly many of these so-called pathological changes in the histological structure of the tissues, to determine whether we really have to do with abnormal morbid conditions.

The conditions found are as follows:—

1. We have a true pathological tissue change; or

2. We have to deal with changes due to the methods of preparation (decomposition, hardening, &c.); or

3. The apparently morbid change is frequently found in tissues quite healthy; or

4. Lastly, we may have to do with histological conditions which are met with so constantly in normal organs, that in a way they may be regarded as characteristic of healthy structure.

I consider it most essential to have some test of the true value of the appearances seen by the microscope; the immense array of material which has been collected must be properly arranged, and many errors must be discarded.

I have, some time back,[1] occupied myself with the relations of the small vessels of the brain, to settle the boundary between the normal and pathological vascular changes. In spite of this, I have very frequently found that the occurrences of vessels considered to be diseased, have afterwards been found present, and never absent, in every healthy brain. It is to be noticed that such a false explanation brings in its train a series of false conclusions, and often in considerations of very great importance.

I must here explain that the above-mentioned work is not generally known to English readers, and I think it advisable to bring before them in these pages some of the points in my work, and the reader will pardon me if I occasionally repeat what has been already published.

It is scarcely necessary for me to refer to the important meaning which pertains to the blood-vessels of the central nervous system. It should be assumed that the nervous elements, and especially the ganglion cells, are more sensitive than any other parts of the tissues to disturbances of their normal nutrition. The anatomical and physiological entirety of the blood-vessels is necessary chiefly for the normal function of the organ of the central nervous system; and therefore it is again evident how very important it is to distinguish between the normal and morbid vascular changes.

In the following pages I shall not take account of the larger vessels, which run outside the brain and spinal cord; but I will confine myself to the smaller vessels, which are already imbedded in the nerve substance.

Methods of investigation.

The best representation of the structure of the smaller vessels of the brain is obtained when they are examined in the

[1] 'Beiträge zur Pathologischen Anatomie der Gehirngefässe,' Wien. Med. Jahrbücher, 1877.

fresh condition, and with the use of as little chemical reagents as possible. I have made it the rule in every case, where a diseased brain is in question, to make examination of the fresh organ, that is of the small vessels, and I can very strongly recommend this practice; for, after hardening has occurred, nothing much can be recognised. Pieces of brain or spinal cord, not too small, must be macerated in a pale yellow solution of bichromate of potash, for 24 hours (2–4 days will not hurt). If the brain-substance be now coarsely separated numerous vessels are found, which can be isolated (always under water) by means of a couple of needles from the surrounding nerve-substance. In this way preparations can readily be made of the entire arterioles with their ramifications, together with the beginning of the capillary network; and it can be so managed that the vessel is scarcely, or not at all, damaged by the needle, if it be held firmly on the surrounding nerve-tissue.

The prepared vessel can now be immediately examined in water, or very weak salt-solution, and thereby most of the true details of the structure can be recognised. If the edge of the cover glass be surrounded with damar-lac, and care be taken to do this thoroughly, the preparations can be kept for a very long time in water (I have kept some for ten years) without any appreciable change (with a few solitary exceptions).

The preparations can be stained by aqueous solutions of various dyes (carmine, picrocarmine, hæmatoxylin, aniline dyes), and after rinsing in water, they are to be preserved in the water as shown above.

The generally used fluids, glycerine, oil of cloves, &c., are to be avoided, as they produce marked changes in the vessels.

Certain pathological conditions—there are only a few—should especially be studied in cut sections; for this purpose the well-known methods of hardening are to be employed.

Structure of the normal vessels of the central nervous system.

1. *Structures of the arteries.*—If we examine the smallest arterial vessels, we can distinguish four coats, which I will

describe, from inside out, as endothelium, membrana fenestrata, tunica muscularis, and adventitial lymph-sheath.

(*a.*) The endothelium is a very delicate membrane (Fig. 1, *a*) consisting of a simple layer of flattened cells; the ground substance between the cells can be demonstrated by the usual method with nitrate of silver. The nuclei of the endothelium-cells, which become apparent after staining with carmine, are an elongated oval in shape, or like a whetstone, and are arranged with their long axes in the direction of the flow of blood. In Fig. 1, where the other layers are present over the endothelium, the cells of the latter are clearly to be made out by the longitudinal arrangement of their nuclei. Nearly always at the edge of the cell-nucleus is seen a strongly refracting bright granule, which projects into the nucleus itself, and the meaning of which is not yet explained.

The endothelium has a tendency to run into folds, when it is detached from the other coats; it can be found best (together with the membrana fenestrata) by teasing with needles one of the intra-cerebral arterioles, after they have been stained with carmine. Th. Deecke[1] has observed, within the just described endothelial flattened cells, yet another layer of cylindrical cells, and he adds further, that the innermost layer of cylindrical epithelium disappears in the smaller branches, "which have entered the brain-substance, and always after their first division."

(*b.*) *The membrana fenestrata*, Fig. 1, *b*, contains no nuclear or cellular constituents, but appears as a glassy, compact, membrane, which always presents very sharp, clear, longitudinal folds. When more highly magnified, numerous small bright spots (or holes?) are to be seen. They are most visible in one of the large arteries, which are very numerous in the basal cerebral ganglia. In the arteries of the convexity of the brain the membrane is extremely delicate, and disappears usually as the calibre of the arterioles diminishes. One of the important layers outside the membrana fenestrata, which Deecke (loc. cit.) described as a "tendon-like mem-

[1] "The Structure of the Vessels of the Nervous Centres in Health, and their Changes in Disease;" 'American Journal of Insanity,' 1877–81.

brane," I have failed to discover,—at least in the smaller arteries.

(*c.*) *Tunica muscularis* (Fig. 1, *c*) consists of spindle-shaped smooth muscle-fibres, which encircle the inner layers of the blood-vessel. The sharply notched edge of the vessel gives a clear representation of the individual muscle-fibres, the long oval nuclei of which are distinctly seen after staining with carmine; they are all arranged transversely, and cross the corresponding nuclei of the endothelium. On the larger arteries are many layers of muscular tissue, placed one over the other, and these determine the thickness of the vessel-wall. The finer arteries possess only a simple layer of muscle-fibres, and moreover their form alters with the diminishing size of the arteries, in such a way that the fibres (and also their nuclei) become shorter, and at the same time broader.

Deecke describes, outside the tunica muscularis, a layer "formed by separate large bundles of elastic fibres." I had already seen these fibres, and have described them in my treatise above quoted. I have explained these, as folds of the next layer, viz. the adventitia. I confess that I have frequently entertained doubts as to the accuracy of my view, without on the other hand being able entirely to accept that of Deecke; this, however, I should by no means oppose, that these questionable fibres are found exclusively in the large arteries, and are not present in the smaller ones, a point which Deecke does not mention.

(*d.*) *The adventitia* (the adventitial lymph-sheath, Fig. 1, *d*). This is a delicate connective-tissue membrane, which, extending very much further than the tunica muscularis, is not attached to it, so that between the two coats there is a freely communicating space. The adventitia contains round or oval nuclei, which frequently have a little protoplasm around them, and in appearance they resemble connective-tissue nuclei. Situated on the adventitia are more or less pigment granules, fat-globules, and fat-granule cells; about these normal appearances I shall soon have to speak further. Concerning the presence of endothelial cells on the adventitia I am not more certain than is Deecke; on the other hand, I must endorse the opinion of this author concerning

the character of the adventitia, that it is a perfectly independent sheath.

In cut sections, particularly from the brains of animals, we see sometimes very clearly, as if appearing from the adventitia, stiff, long-drawn-out processes, which project into the brain-substance, and end in the connective-tissue cells. In fresh-teased preparations there is nothing to be seen of these numerous processes; I must confess that it does not seem to me quite clear how, in preparations made as delicately as possible, all these fibres, or the greatest part, can be torn away from the adventitia.

For the most part, I incline to the opinion that, outside the adventitia, a delicate connective-tissue membrane exists, somewhat like an inner coating to the canal in which the vessel courses.[1] This outer sheath of the vessel, derived from the pia mater, is so intimately connected by the numerous above-mentioned processes with the connective tissue present in the interior of the brain-substance, that, even with the most delicate manipulation, this membrane remains behind, while the whole vessel with its adventitia is detached. In a hardened preparation the outer sheath is so closely applied to the adventitia, and this is so crumpled up with the inner vessel-coats, that it can no longer be recognised as an independent membrane.

I scarcely need affirm, that the adventitial vessel-sheath is in no way intimately fused with the brain-substance which forms the ultimate external sheath; there remains a space between the two, which frequently appears to be completely reduced to a minimum. We have outside, as well as inside, the adventitia a space where fluid can circulate; under normal conditions we cannot speak of any fluid, besides the animal juices, which are set apart to bring about the change of matter between the blood and the tissue of the organ; we can designate this juice shortly as "lymph," and from this we can speak of two different lymph-spaces, one between the tunica muscularis and adventitia (adventitial lymph space, *Virchow, Robin*), while the other is outside the adventitia (perivascular lymph-space, *His*). In

[1] Compare Adler, 'Archiv für Psychiatrie,' 5 B.

both named lymph-spaces, under certain conditions, we find, more or less, numerous lymphoid corpuscles; but whether they are, as Deecke thinks, only wandering white blood-corpuscles, it is not easy to decide.

I should like to remark, with regard to the perivascular lymph-space, that it should be looked upon as a cavity between the tissues, and that it communicates with numerous other cavities, which serve to bring about the circulation of the nutrient fluid to the tissue elements. I have shown[1] that the ganglion cells of the grey substance are situated in a hollow space, "pericellular lymph-space," which under certain conditions may be very small, but it is always present—small though it be—between the protoplasm of the nerve-cells and the neuroglia. These pericellular spaces are in communication with the perivascular spaces, and are certainly just as little an artificial product as the latter. The proofs of this can be found in both my above-quoted works, as well as in another essay.[2]

2. *Structure of the veins.*—In the intracerebral veins I can distinguish only three layers.

(*a.*) *The endothelium* is much more irregular than that of the arteries, the cell-nuclei are for the most part rounder, not so elongated and less symmetrical, and are situated in a position corresponding to the longitudinal direction of the vessel.

(*b.*) *For the middle coat,* we must take that tissue which forms the principal part of the wall of the vein; it consists of connective tissue with scattered round nuclei, and only a few irregularly arranged muscular fibres.

(*c.*) *The adventitial lymph-sheath* corresponds with the description of the arteries given above, with a single difference, about which I will speak later.

I may mention that *Deecke* gives a description of the structure of the cerebral veins, differing from mine in many points.

3. *Structure of the capillaries.*—In these, not more than two layers can be seen.

[1] 'Ueber einige Lymphräume im Gehirne,' Sitzungsber. der K. Akad. der Wissensch. zu Wien, 61 B.

[2] 'Ueber Ektasien der Lymphgefässe des Gehirns,' Virchow's 'Archiv,' 55 B.

The capillary tube proper is formed from a continuation of the arterial and venous endothelial membranes, which have been divested of their outer coats, only the adventitial sheath being visible in many places; I hold the opinion that the adventitia forms round the arteries and veins, as well as all the intracerebral capillary system, an uninterrupted sheath; only over the capillaries this is finer and narrower, so that it often escapes observation.

Further peculiarities of the structure of the adventitial sheath of the brain-vessels.

I have mentioned earlier, that in the adventitia pigment and fat are found; on this point I must make some further observations.

When we look at the central nervous system of new-born children, we find numerous fat-granule cells; it is highly manifest that these play a considerable part in the development of the medullated fibres; they convey the material for the formation of the medullated sheath, just as, by morbid dissolution of the medullated fibres, the fat-granule cells again appear, and take up matter they come in contact with and remove it.

Similar fat-granule cells are found again in the new-born child, in the midst of the whole nervous system, and not a few are firmly attached outside the adventitia of the vessels; the flow of lymph is quite incompetent to tear them away, and so they remain like "stranded waifs," when all the other fat-granules have disappeared from the central nervous system, the development of the medullated fibres being completed.

If we now examine some of the vessels (artery or vein) from the brain of a child under two years old, we shall find, perhaps not on every vessel, still very frequently, these already-described fat-granules, which under a low magnifying power stand out very clearly as dark specks. We shall certainly find, after a short search, in every child's brain such vessels with fat-granules on the adventitia.

We will now examine an adult brain. On the adventitia of the veins fat is constantly found; I have found it without

exception in every brain. At the least, we find in places single drops of fat, but nearly always evident fat-granule cells. The latter is present in great number, and give to the vessel a peculiar spotted appearance; not uncommonly the fat-granules form so numerous patches, that they surround the vessel for a larger or shorter piece with a perfect ring of fat (compare Fig. 2); this can be observed best in the smaller veins; but in the capillary vessels themselves fat-granules are by no means rare; their presence is a further proof that the capillaries possess their own lymph-sheath. I must here emphasise that these granule cells of the adventitia are situated always on the outside, accordingly they form a corresponding projection, as we see plainly in Fig. 2.

It may be interesting to note, that I have also found a considerable amount of fat in the cerebral veins of very emaciated subjects.

From the circumstances that these fat-granule cells are found in the adventitia of the cerebral veins in the new-born condition as well as in old age, and are seen in variable quantities after deaths from different causes (even after sudden and most violent death), I am justified in stating that the collection of fat on the adventitia of the cerebral veins, even when very abundant, is usually not to be considered a pathological occurrence; we see in these drops of fat and fat-granules (at least most of them) the remains of the period of embryonal development.

In case the fat-granules should be found in pathological changes of the central nervous system (as softening of the brain, &c.), they will be seen in great quantities on the vessels, vein and arteries. So, for example, we frequently observe in the spinal cord with degeneration of the posterior columns, even with the naked eye, the vessels which pass into the posterior commissure with their lateral branches presenting a thick white appearance. In such a case, where the surrounding nerve-tissue is diseased, the fat-granule cells on the adventitia are self-evident, as the expression of a pathological process, which attacks the vessels only secondarily.

In yet another way fat-globules can make their appearance on the adventitia (veins or arteries). It appears then on the

inner side of the adventitia, which is clouded by a fine precipitate of very small drops of fat. This occurrence is rather rare, and appears to me to be the outcome of a morbid disturbance of nutrition.

We have spoken hitherto chiefly about the veins. When we prepare an *artery* from an adult brain, we find frequently, but not invariably, fat as in the adventitia of veins. On the other hand, the presence of pigment is constantly observed. This pigment, consisting of smaller or larger round globules, varying from light yellow to light brown, is found on the outer surface of the adventitia; frequently several of these granules are united with others to form aggregations (Fig. 1, *e*). In young children of two, three, or more years old, this pigment is very light yellow, or almost colourless, and shows all transitions up to the colourless fat-globules, mixed with which the pigment-granules are frequently found. This pigment hardly changes colour at all under the action of acids and alkalies; with osmic acid it takes a deeper colour and assumes rather a greyish tint; this reaction is more marked, the brighter the pigment has been before.

In my work on the cerebral vessels I have given a tabulated description of the condition of these vessels in ninety-one brains, and, with the exception of children under two years of age, I have never found pigment absent from the adventitia of the arteries. The relative quantity of pigment is such, that it can vary within certain limits, but it is perfectly independent of the causes of death, and cannot be referred to any morbid change in the individual in question.

I must particularise this point, for this pigment is always erroneously regarded as the effect of severe congestion of the brain, or even of actual hæmorrhage; it is confounded with the colouring matter of blood, which may also be found here, and I shall soon have much to say on this subject.

The question now arises, whence comes this pigment, which is found quite normally in the arterial adventitia of every brain, with the exception of young children?

I hold the opinion that this pigment arises from those fat-globules, which in early life are left behind on the adventitia of the cerebral vessels. Perhaps it represents a higher stage

of oxidation of this fat, and arises by some change of tissue, through combustion of the fat.

Therefore we can speak of the following conditions:—

1. In very young subjects a very bright pigment is found, which resembles fat-granules in form and colour, and shows a stage of transition to the darker granules.

2. We often see on the arteries considerable collections, where fat and pigment are mixed together; also we nearly always find, in the same place, all the transitions from colourless fat-globules to yellow-brown granules.

3. The dark colour produced in the light-tinted pigment granules by the action of osmic acid, reminds us of the dark grey colour which fat receives from this reagent.

4. This pigment is also found on the veins; its proper situation, however, is on the adventitia of the arteries; in the arteries circulates blood rich in oxygen, here a more active change of tissue prevails, and on that account the change from fat into pigment has its seat here.

I must here add, to prevent misunderstanding, that I do not consider that the collections of fat found in the infantile brain must still be met with in the adult brain, as fat or pigment; a great part is perhaps absorbed in the mean time.

I will now take the opportunity of mentioning, yet another condition under which pigment may be found on the adventitia of the brain-vessels. True pigment cells are very frequently found on the delicate membranes of the brain (viz. on the medulla oblongata and base of brain). These cells are longish, for the most part with many thick processes, and with a nucleus which generally stands out bright in the midst of the finely granulated dark-brown pigment. This pigment is also seen on the larger vessels which pass into the base of the brain, they are merely introduced from the meninges. Not the least pathological significance can be attached to them.

Forms of pathological pigment on the cerebral vessels.

We have just seen that pigment is normally found on the brain-vessels under two forms.

There are deposits of pigment which can only be regarded as pathological, and particularly

1. *Blood-colouring matter, Hæmatoidin.*—This is seen as diffuse colouring matter of round granulated cells (fat-granule cells, or lymph-corpuscles), or in the form of larger or smaller red-brown bright granules; not uncommonly we find hæmatoidin crystals in the shape of rhombic columns. Such crystals or amorphous granules are frequently seen in the interior of the fat-granule cells. In isolated vessels we find the blood-colouring matter either applied to the adventitia or situated in its lymph-spaces; in cut sections we see that also the surrounding nerve tissue is nearly always richly pervaded with this pigment, namely with cells containing pigment. The presence of a large quantity of this pigment is always a sign that, in this place, an exsudation of blood has occurred.

The formation of hæmatoidin crystals as well as the diffuse yellow-coloured cells is easily recognisable; on the contrary, the granule-forms cannot be distinguished from normal pigment without further investigation. We possess a reaction which permits us, even after hardening in the chromate salts, to make out this difference without a doubt. The blood-colouring matter, under the influence of concentrated sulphuric acid, passes through a series of colours, viz. green, blue, and violet, and is finally discharged. Normal pigment hardly changes at all when thus treated.

2. *Malarial Pigment.*—In people who have suffered from attacks of intermittent fever the brain shows an almost dark grey colour, which is easily recognisable. This colour proceeds from a dark, nearly black colouring-matter; it represents granules which vary from the smallest dimensions to the size of a red blood-corpuscle.

Next we find these dark granules only in the blood, partly free in the plasma, partly in the interior of the blood-corpuscles. A considerable part of these pigment-granules can pass from the blood into the walls of the vessels, and fix themselves there; on the other hand, we do not find this intermittent pigment in the rest of the central nervous tissues.

Larger aggregations of this pigment can easily block one

of the smallest vessels (pigment-emboli), whereby a rupture of the wall of the vessels ensues, and a small hæmorrhage occurs. This pigment is easily recognised by its dark colour, by its capability of resisting very different reagents, and above all by its presence in the blood itself.

We have therefore learnt to distinguish two kinds of normal and two kinds of pathological pigment on the vessels; I will presently bring forward an artificial pigment which has only had a short duration of existence. *Erlitzky* recommends as a hardening fluid a solution of bichromate of potash with sulphate of copper; this mixture found favour very quickly; and *Erlitzky* soon after described a spinal cord, which was hardened in his fluid, and pretends to have shown a very remarkable pigmentation (viz. in the vessels). I had originally a very doubtful opinion of this pigment,[1] but as a short time ago I saw a preparation of this kind, and have myself made observations with hardening in this fluid, I cannot have the least doubt about it that it is a precipitate, artificially caused by the hardening fluid. Since then, this appearance has been mentioned by others (W. v. Tschisch).

I take this opportunity of giving an example, how different artificial productions can be raised to the dignity of pathological formations, and I am justified in taking this example because *Erlitzky's* fluid appears to be generally used, and I would give a warning against further errors which might be produced by the action of this fluid.

Some histological changes of the brain-vessels, which occur under normal conditions.

After examining numerous cerebral vessels, I am convinced that there are in them a number of histological abnormalities, which, although they are considered to be pathological, are still met with in brains which, being in all other respects both anatomically and functionally healthy, must be considered quite normal. I will further add, that the changes presently to be described in the vessels need not produce any damaging influence, nor do they bring about in the living state any

[1] Compare 'Centralblatt für Nervenheilk.' 1881, pp. 399–407.

disturbance of function, or in the dead subject any anatomical lesion which can be connected with them.

This point is of great importance, from the fact that we should be forewarned of any judgment we may make from what we find by the microscope; we should not therefore, without further investigation, draw any conclusions from the presence of such vessels of any disturbance in the function of the brain.

1. *Deposit of calcareous matter on the vessels.*—Calcareous deposit (especially as carbonate of lime) can take place on the adventitia, as well as on the tunica muscularis.

In normal brains we do not often find chalk on the adventitia. If the calcareous deposit starts from the adventitia, it seldom remains limited to this membrane, generally the muscularis will be involved in the morbid change; but on the other hand the calcareous deposit can grow from the outside, as roundish or club-shaped chalk masses, which may attain a considerable size.

On the muscular coat I have frequently found chalk in perfectly healthy young subjects (viz. in girls 8 and 19 years old).

At first we see masses surrounding the muscular coat, with a peculiar slightly shining appearance; in further stages the whole muscular coat of an artery is changed through a longer portion of its course into a stiff finely granulated tube (Fig. 3). Even by macroscopical examination such a vessel can be seen by its white colour and needle-like rigidity. On moving it with a needle, the resistance is easily felt. Under the microscope we can recognise places where the calcareous cylinder is broken during preparation, and it shows cracks like broken glass. We are concerned here for the most part with carbonate of lime, and we can use in addition the reaction with sulphuric acid to make the diagnosis certain. By the reaction of this acid, bubbles of carbonic acid are formed, and fine needle-shaped crystals of calcic sulphate.

If the calcification is further advanced, we see the whole capillary network presenting a streaked appearance and changed into fine calcareous tubes. In the cerebellum, and especially in the granule layer of its cortex, such diffuse

calcification can be repeatedly found, but it is not easy to say with certainty whether the adventitia or the muscular coat is the commencing point of the process.

We must suppose, *à priori*, that a calcification of the muscular coat will not only damage the nutrition of the brain, but, owing to the brittle condition of the vessels, will lead to further danger.

On the other hand I can state from experience, that with the exception of the higher stages of disease, such calcification, as has been mentioned, occurs very frequently under normal conditions; but hitherto, contrary to my expectations, I have not found any calcification of the muscular coat in cerebral hæmorrhage.

Calcification of the tunica media, if not very much advanced, has hardly any pathological significance.

2. *Fatty degeneration of the muscular coat.*—In preparations such arteries appear to the naked eye of an opaque yellow-white colour, without that rigidity which is found with calcification. Under the microscope we see the muscular layer filled with very small point-like fat-granules, and we can only here and there stain the muscle-nuclei slightly with carmine. Such a vessel shows under the microscope great similarity with a calcified one; except that the fatty muscular sheath is more yellow in colour, and never shows the fissures and cracks characteristic of the calcification; lastly, the sulphuric acid reaction is absent. This degeneration of the muscular coat we must also consider as a process which leads to serious functional impairment of a very important arterial covering.

In spite of this, I have met with this fatty change in healthy as often as in diseased brains, in old as well as in young subjects, and in those who have succumbed to acute as well as chronic diseases.

We cannot therefore attach great pathological meaning to this fatty change of the muscular coat.

3. *Connective tissue degeneration of the vessels.*—In arteries as well as in veins (the latter more frequently) an overgrowth of connective tissue proceeding from the middle coat (tunica media) of the artery takes place. The lumen of the tube is at first unaffected, while the circumference

of the vessels increases; thereby also the adventitia may remain intact. So we find pretty frequently spindle-shaped swellings of this nature on the little arteries or veins. It seems to me almost certain, that these enlargements of the vessels can usually be referred to the peculiar conditions of the blood-pressure which prevails at those places where a sudden change occurs in the calibre of the vessels; on this account they are not found so often at those places where the vessel divides dichotomously, but usually where the smallest vessels branch off immediately from relatively large trunks. A favourite place for finding the degenerated vessels is the highest layers of the cerebral cortex. *Neelsen* has shown,[1] that at this place these changed vessels are to be found in most brains, and I have also had the opportunity of convincing myself of this discovery.

Commonly we find, near the above-mentioned places, vessels in which the degeneration has made further progress. The connective-tissue overgrowth reaches at last the periphery of the vessel-lumen, the vessel is obliterated and continues its course as a cord of fibrous tissue. I have very frequently met with such vessels in perfectly healthy brains, even in children eight years old, but I must add, that this degeneration-process occurs much oftener and in a more marked degree in the vessels of old age (over sixty years), when once atrophy of the brain has commenced.

Although the blocking of the vessels caused by the fibrous overgrowth occurs gradually, it appears wonderful that a process by which individual regions—small though they be—of the brain are robbed of their nutritive arteries can be referred to a normal occurrence, and produce no known disturbance.

But on this point I should express myself with more caution. There are many obscure transitions in the domain of our cerebral life, some or other of which may perhaps be explained by local disturbances of nutrition, which comes about through such a blocking of vessels.

Deecke (loc. cit.) has confirmed the frequency of this change, for which he has used the expression "callous degeneration,"

[1] 'Archiv f. Heilk.' xvii., B.

and calls it a process situated "at the border of physiological and pathological conditions."

4. *Dilatation of the adventitial lymph-sheath.*—Chiefly on arteries we occasionally see in places the adventitia, bulging like a blown-out bag, and separated some distance from the vessel. Such partial dilatations are seen in small as well as in large arteries. Fat-granule cells and pigment granules are often collected on such sacs.

Since such dilatations are often found in healthy brains, they must be considered as normal conditions.

If these bulgings of the lymph-sheath become very considerable and extending over numerous vessels, they must then be considered pathological.

More extended dilatations of the adventitia lead to the formation of cysts, which are visible to the naked eye. I have described the appearance of such cysts on the Pons Varolii, and they may be considered as cysts due to retention of fluid; the adventitial lymph-chamber is blocked by elements which have arrived in the lymph stream; whereby a stoppage of this lymph stream is produced, and hence the space becomes distended.

If the distension of the adventitia is diffused over many vessels and through a considerable tract of their course, the brain acquires, on cross-section visible to the naked eye, a peculiar sieve-like appearance studded with holes, a condition called "état criblé." This "état criblé" can arise through the enlargement of the perivascular lymph-spaces, as for instance through atrophic shrinking of the brain-substance; in this condition the adventitia, as well as the other coats of the vessels, remains unaltered.

The "état criblé" is chiefly confined to the white substance, whilst the cyst-formations as described above are only found in the grey matter.

Some important changes in the vessels which are decidedly pathological.

In the literature of this subject there are a very great many pathological changes of the brain-vessels which are certainly of very great interest.

I will, however, in the following pages only mention a few morbid appearances which seem to me of very great importance, purposely omitting the rarer points.

1. *The atheromatous fatty degeneration of the intima.*—This morbid process becomes evident by the presence of larger or smaller dark spots on the inside of the vessel (Fig. 5). Outside these spots we can readily recognise the healthy muscular layer; by pressure on the cover-glass it not unfrequently happens that these atheromatous masses are set free, float along further with the contents of the vessels, and finally become fixed on a narrow part of them.

We can also follow the formation of emboli in a small vessel from the point where it is liberated from the intima to where it blocks up a small branch.

By a higher magnifying power we see that these above-described deposits in the intima consist of a granulated amorphous mass, generally with very numerous fat-granules.

In nearly all cases of cerebral hæmorrhage I have been able to find this change in the intima, and this is particularly evident in the neighbourhood of the hæmorrhagic focus. I have not the least doubt that a great many of these cerebral hæmorrhages can be referred to this morbid change in the vessels, and especially that these atheromatous masses are torn from the intima to form emboli in the smaller branches.

We can also find this degeneration of the intima without any cerebral hæmorrhage, but this proves nothing against my view, as the atheromatous disease of the vessel must first be present, and then follows the embolism and hæmorrhage. I should therefore like to define the fatty-atheromatous degeneration in the intima as one of the most serious diseases of the smaller brain-vessels.

2. *The paralytic dilatation of the small brain-arteries.*—I have frequently found in brains, in chronic mental diseases, numerous arteries changed in the manner shown in Fig. 4. The muscular coat does not show a tube of even calibre, as it normally should, but it presents a succession of irregular, but not excessive, dilatations and constrictions; no other morbid changes can be recognised on the muscle-fibres,

even by using stronger magnifying powers. Most frequently I have found this condition of the arteries in cases of general paralysis. Later on, a chronic disturbance of the innervation of the brain-vessels may with great certainty be assumed to be caused by this disease, and I explain the above-described appearance as follows: that a partial paresis of the individual groups of the muscle-fibres in the tunica muscularis, or at least a diminution of the muscle-tonus, which after some time is produced, gives rise to these diffused enlargements of the vessels, which become permanent, and can be discovered after death. In the absence of any disease which can by other means be recognised, it seems to me that this explanation corresponds best to the conditions found.

This partial enlargement of the small arteries would therefore be considered as a sign of a chronic vaso-motor paralysis.

3. *Abnormal products contained in the lymph-space.*—Under normal conditions we find only a few formed elements in the adventitial lymph-space. We see single lymph-corpuscles, less frequently fat-granules, pigment granules, and rather large vesicular cells, which must be looked upon as metamorphosed lymph-corpuscles; exceptionally we chance to see a red blood-corpuscle. If the cover-glass be pressed upon, the above elements found between the muscular and adventitial coats float further on.

If we find numerous red blood-corpuscles in the adventitial lymph-space, their presence there is exceptionally due to diapedesis; more commonly it happens that a rupture has occurred in the inner coats of the vessel, and we have to do with a dissecting aneurism.

Very often we find an increase in the lymph-corpuscles in the adventitial lymph-space, their quantity can be so considerable that the muscular coat is so covered up with many layers of these elements as scarcely to be recognised. I consider that this increase of the lymph-corpuscles (improperly called "nuclei-growths") is to be referred to an emigration of white blood-corpuscles; we find it in different hyperæmic and inflammatory conditions of the brain. Also on the brain-vessels of general paralysis cases this increase is very frequent, as I

have shown;[1] and just at this place the tendency to migration of the lymphoid elements is very remarkable.

Hitherto I have considered only the pathological increase of these elements, an occurrence which can also take place normally in the adventitial lymph-space; but we find there sometimes many kinds of pathological products, the presence of which shows a morbid condition. In purulent meningitis we can generally trace the pus-corpuscles along the vessel-sheath some distance into the interior of the brain; whether these corpuscles have had their origin on the spot, or have merely migrated out of the pia, it is frequently difficult or quite impossible to decide.

From a special pathological point of view, it seems to me that different kinds of neoplastic elements can be found in the adventitial lymph-space; I have seen there, cells of tubercular, sarcomatous, cancerous and syphilitic new growths. Such cells can be found easiest in the neighbourhood of a corresponding tumour. We may therefore conclude that the lymph-tracts of the brain afford a very convenient path for diffusing the various new growths, and the increase of a tumour often occurs through the intervention of this lymph-tract. In inflammatory processes, especially in the spinal cord, and also in lyssa, I have found round the arteries, in the adventitial and perivascular lymph-spaces, a peculiar regular-shaped substance without any recognisable structure, but colouring strongly with carmine. I believe that we must recognise in this substance the coagulation-product of a transudation proceeding from the blood.

I pass over a great number of changes of a different sort, which are to be found on the smaller brain-vessels.

The task which I have set myself to do has not consisted in giving a complete enumeration of all the diseases of the brain-vessels. I wish above all things to point out that much, which has passed current with us as morbid, is still within the confines of a normal state; with this I have thought it desirable to associate the discussion of some purely pathological

[1] 'Zur pathologischen Anatomie der Dementia Paralytica,' Virchow's 'Archiv,' 52 B.

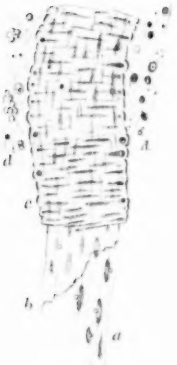

Fig. 1.

Fig. 2.

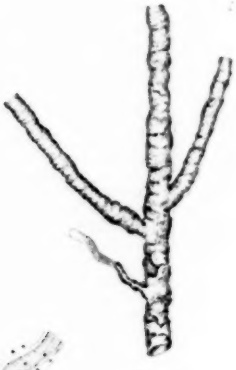

Fig. 3.

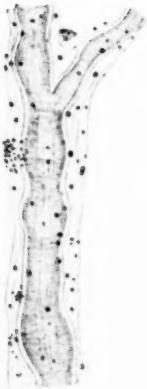

Fig. 4.

Fig. 5.

conditions, which seemed to me of great importance from a scientific point of view.

EXPLANATION OF THE PLATE.

1. A small artery of the brain, torn in such a manner that the individual layers are visible.
a. Endothelial membrane.
b. Fenestrated membrane.
c. Muscular coat.
d. Adventitial lymph-sheath.
e. Little collections of pigment.

2. A normal artery, with fat-granule cells on the adventitia; these form in patches a perfect ring round the lateral twigs.

3. An artery in which the muscular coat is perfectly calcified, at the lower end of the chief branch the calcified tube becomes fractured.

4. An artery from the brain of a patient who died from general paralysis of the insane; we see, scattered about, little enlargements caused by the paralysis of the muscular coat.

5. An artery from the neighbourhood of an apoplectic focus; the intima has fatty-atheromatous degeneration, while the muscular and adventitial coats are intact.

(*Translated from original manuscript by* C. E. Beevor, M.D.)

ON THE EARLY OCCURRENCE OF ANKLE-CLONUS IN HEMIPLEGIA.

BY A. PITRES, M.D.,

Professor of Medicine to the Faculty of Bordeaux.

THE occurrence of ankle-clonus in hemiplegia of ancient date is a phenomenon both frequent and easy to demonstrate. Its semeiological and prognostic value has been shown by the researches of Professor Charcot, and we know that it usually coexists with late rigidity of the paralysed limbs and exaggeration of the knee-jerk. But accurate data concerning the exact time at which it may show itself after the apoplectic stroke are still wanting.

Professor Charcot has shown that in most cases the ankle-clonus precedes by a few days or weeks the appearance of secondary contractures (late rigidity). In a patient in his wards "ankle-clonus could be elicited eight days after the attack, and it was only a fortnight later—that is to say after a lapse of about three weeks—that late rigidity began to appear. In another case the foot-clonus became apparent only a month after the stroke, and rigidity supervened towards the end of the second month."[1]

Professor Charcot gives no further details on the point now under discussion. Professor Westphal is somewhat more explicit. According to him, ankle-clonus usually appears between the seventh and twenty-first day after the hemiplegic onslaught; and cannot be demonstrated immediately after the attack, nor during the next few days.[2]

[1] Charcot, 'Leçons sur les Localisations dans les maladies du Cerveau.' Paris, 1876, p. 301.

[2] Westphal, 'Ueber einige Bewegungserscheinungen an gelähmten Gliedern.' 'Archiv für Psychiatrie und Nervenkrankheiten,' vol. v., 1875, p. 810.

From investigations to which I have submitted a certain number of hemiplegics admitted into my wards in the Hôpital Saint-André a few hours or a few days after the stroke, it results that the clonus may appear at a much more early date than Professor Westphal believes. In most cases, it is true, the phenomenon becomes observable only between the second and the sixth week; but it is by no means rare to witness it within the next few hours after the apoplectic seizure. Thus, in the two cases briefly recorded here, ankle-clonus was noticed eleven and fifteen hours respectively from the beginning.

CASE I.—*Cerebral lesion, followed with persisting hemiplegia. Ankle-clonus observed eleven hours after the accident.*

Mr. X., a law student, aged 22, was brought to the Hôpital Saint-André on the 4th of July, 1883, at 5 in the morning. He had received three-quarters of an hour previously, in a duel, a sword-thrust which penetrated into the right orbit. Immediately after being wounded he fell unconscious. Ten minutes afterwards he came to himself again, and from this moment a complete hemiplegia of the face and limbs on the left side was noticeable.

The regular examination of the patient was made at 3 P.M., eleven hours after the accident. Mr. X. is at this time in full possession of his faculties. He answers with precision to the questions addressed to him. The right superior eyelid is the seat of a marked ecchymotic tumefaction. The sword penetrated, outside the eyeball which was not perforated, at the level of the external cul-de-sac of the oculo-palpebral conjunctiva. It is uncertain how deep it went into the skull; the point was soiled with blood on a length of about five centimetres.

The left hemiplegia is total and complete. The mouth is deviated to the right, and is immovable on the left when the patient speaks. The limbs on the left side are inert and flaccid; they may be passively moved in every direction without showing any trace of rigidity. Voluntary movements are absolutely impossible. Sensation to pricking is diminished. The muscular sense is lost. When the eyes are closed, the

patient cannot realise the position given to his limbs. Motion and sensation are normal on the right side.

The knee-jerk is much exaggerated on the left, slightly on the right. When the toes of the left foot are slightly raised a marked clonus ensues; the individual jerks are accentuated, and persist as long as the dorsal flexion is maintained. On the right side only two or three slight oscillations are obtained, which cease spontaneously though the foot be held firmly flexed.

From the 5th to the 7th of July the tendon phenomena remain the same as on the first day. No modification in the paralysis; no trace of rigidity.

During the next few days movement appears again in the left limbs, without any modification in the knee-jerks. The clonus when elicited on the left side remains the same; but it disappears on the right.

On the 16th, Mr. X. can stand and take a few tottering steps. He leaves the hospital.—We see him again on the 2nd of August and the 28th of October following. There is a rapid restoration of power in the left limbs. There is no appreciable rigidity, but the knee-jerk on that side is still much exaggerated, and the ankle-clonus as lively as on the first day.

On the 8th of July, 1884, we notice that the facial paralysis has almost entirely disappeared. Movements are possible with the left hand, but they are slow, clumsy, and lacking firmness. Muscular sense and tactile sensibility are profoundly altered; with the eyes closed Mr. X. does not realise the position given to his left hand, nor recognise a pencil or coin placed between his fingers. There is some permanent rigidity in all the articulations of the upper extremity.

The left leg is the seat of considerable rigidity. The dorsal flexion of the left foot causes a strong and persistent clonic trepidation, which is not observed on the right side. The left knee-jerk is much exaggerated; the right almost normal.

Dynamometric state of force

right arm,	53	kilogrammes.	
left arm,	36	„	„
right leg,	43	„	„
left leg	21	„	„

CASE II.—*Right total and complete hemiplegia. Right ankle-clonus observed fifteen hours after the initial seizure.*

P. B., aged 66, had an apoplectic stroke with unconsciousness the 4th of June, 1883, at 2 A.M. He was taken to the hospital the same day at 5 P.M., fifteen hours therefore after the seizure. On entering the ward, patient seems still confused; he talks with difficulty, but can yet answer the questions addressed to him.

Right total and complete hemiplegia, without rigidity. The limbs are inert and flaccid. They may be moved in every direction without offering the least resistance; when lifted and left to themselves, they fall heavily on the bed. The right knee-jerk is much exaggerated, the left not so much. When the right foot is raised by the toes, a series of sudden and lively contractions is produced; but after six or eight oscillations this clonus ceases spontaneously, though the foot is kept dorsally flexed. No clonus on the left side.

June 5th.—Patient remains in the same state.

June 9th.—Clonus appears to be more easily elicited than on the first day. The oscillations are longer, and fourteen are counted before they cease spontaneously. No rigidity.

The patient returns to his home on the 10th. I see him a year afterwards: the hemiplegia has persisted, total and complete. The limbs on the right side are the seat of a marked late rigidity. Knee-jerk brisk and powerful on the right, normal on the left. Ankle-clonus very easily obtained on the right, and persisting as long as the foot is held up.

I have obtained evidence of ankle-clonus on several other patients admitted into the hospital from the second to the seventh day after the hemiplegic attack; but it is unnecessary to bring forward the observations in detail. The two cases just described suffice to show that the phenomenon is not always a late one, but may, on the contrary, appear a few hours after the seizure, at a period when secondary degenerations in the cord have not yet had time to be developed.

I shall add a few words concerning the prognostic value of the precocious ankle-clonus. I have hitherto seen only one case in which a patient presenting this sign recovered. It was that of a man, of 33 years of age and syphilitic, who, after having suffered from persistent headache, became hemiplegic on the night of the 18th to the 19th of December, 1882. He was admitted into the hospital thirty-five hours after the attack. He then had a total right hemiplegia with exaggerated knee-jerk on that side. Ankle-clonus was easily elicited in the right foot. An energetic antisyphilitic treatment was at once instituted. Ten days after, the clonus had disappeared, and the paralytical condition had already improved to a marked degree. Soon after, the patient left the hospital quite well.

With this exception all the other cases in which early ankle-clonus was observed have remained permanently and more or less completely hemiplegic, and with late rigidity of the affected leg.

It would thus seem, that the elicitation of ankle-clonus during the first few hours or days after the apoplectic attack is a symptom of evil omen, and an almost certain sign of an incurable paralysis.

CASES OF TUMOUR OF THE CORPUS CALLOSUM.

BY JOHN S. BRISTOWE, M.D., LL.D., F.R.S.

TUMOURS of the corpus callosum, so far as I know, are rare. But it has happened to me within the last three or four years to have under my care three cases of primary tumour of this organ, and another case in which it became involved in a growth springing from the floor of one of the lateral ventricles. Consequently it has seemed to me worth while to collect these cases; and to consider how far, if at all, the symptoms presented by them were distinctive.

The first case was that of a middle-aged man, who first complained of headache about fourteen weeks before his death. Shortly afterwards his right arm, and subsequently his leg, got weak; and his intelligence became impaired. The symptoms slowly increased upon him, the paralysis of the arm and leg became aggravated; indications of feebleness in the facial muscles appeared; he became apparently more and more stupid and extremely drowsy, and probably to a large extent on this account he ceased to speak, and allowed food to collect and lie in his mouth. Before death he passed into a state of complete coma. There were no fits, no actual sickness, and no affection of the ocular muscles. The chief phenomena, in fact, were headache, stupidity, and drowsiness, in association with paralysis of the arm and leg of one side, and doubtful paresis of the facial muscles of one or both sides—the symptoms being slowly progressive.

At the post-mortem examination a spongy, probably sarcomatous, growth was found to involve the corpus callosum, mainly in its anterior part, and to extend thence on either side into the centrum ovale, the left side being chiefly affected. There was no disease of the grey matter of the convolutions, or of the ganglia on the floor of the ventricles.

The second case also was that of a middle-aged man, whose illness came on while he was at sea, about ten weeks before it proved fatal. It is said that at first his speech became affected and his manner dull. When admitted, four weeks later, he had pretty complete right-sided hemiplegia, with involvement of the face and tongue, and well-marked aphasia. It was thought, indeed, that his symptoms were due either to hæmorrhage into the left side of the brain, or to plugging of the left middle cerebral artery. But the discovery of optic neuritis rendered it more probable that he was suffering from a cerebral tumour; and this view was confirmed by the slowly progressive aggravation of his symptoms. He gradually became absolutely speechless, and, though able to masticate and swallow, unable apparently to protrude his tongue, or to prevent the saliva dribbling from his mouth. He grew more and more dull and stupid, but not specially drowsy; and, though apparently not understanding anything that was said to him, stared intently and steadily at me, or possibly others about his bed. Before his death his pupils became unequal, rhythmical tremors attacked his arms, and his temperature rose.

This patient, like the other, complained of headache; but, also like the other, he never had a fit, did not suffer from sickness, and had no trace of paralysis of the ocular muscles. The chief symptoms were headache, right-sided paralysis, aphasia and dementia. Drowsiness was not specially noted in this case.

After death, a tumour was discovered, occupying and evidently originating in the anterior half of the corpus callosum, whence it extended into either centrum ovale. The left centrum was more extensively involved than the other, and on this side the grey matter of the second and third frontal convolutions and of the ascending parietal was involved. There was no disease elsewhere.

The third case occurred quite recently. This case was that of a middle-aged man. The history is not very clear; but he cannot have been ill altogether more than twelve weeks; and was probably ill for a much shorter period. When received into the hospital ten days before his death, he was partially paralysed in all his extremities, but mainly in the left arm and

leg; there was doubtful paralysis on the left side of the face; he was very drowsy, and his speech was slow and indistinct, but he was sensible. During the progress of the case, the paralysis increased, mainly on the left side, and it was thought also that there was some weakness on the left side of the face; he became profoundly sleepy; he lost the power of speech; he manifested some difficulty in chewing and swallowing; and when awake he, like the last patient, stared intently but with a puzzled look at those about him.

This patient resembled the others in the absence of fits, sickness, and paralysis of the ocular muscles. His chief symptoms were, paralysis of the limbs, drowsiness, stupidity, loss of power of speech, and difficulty in getting rid of food placed in his mouth.

The post-mortem examination revealed the presence of a sarcomatous growth, infiltrating the anterior two-thirds of the fornix and corpus callosum, and extending from the latter into the white matter of both hemispheres, chiefly, however, into the right. Neither the grey matter of the surface nor the ganglia were involved.

The close resemblance, if not identity, between these cases as regards their morbid anatomy is obvious. In all of them the growth had clearly originated in the corpus callosum, and had thence extended almost symmetrically along the radiating fibres of this body into the white matter of the hemispheres. There is reason, indeed, to suspect them to be examples of a class of cases which, though attention has not been specially directed to them, are really not uncommon, and not unlikely therefore to be met with in practice.

There was also a considerable resemblance in the collective symptoms presented by them; and I am inclined to think these were sufficiently characteristic to suggest a correct diagnosis. At any rate my former experience enabled me, when the last case came under treatment, to make a happy guess as to its nature, and accurately to predict, as the corpse lay on the table, what would be found when the head was opened.

The chief characteristic features of the cases were:—1st, their ingravescent character, a character which they possessed in

common with other cases of cerebral tumour; 2nd, the gradual coming on of hemiplegia, for the most part resembling in its distribution the paralytic symptoms usually attending hæmorrhage into one of the hemispheres, or softening due to embolism; 3rd, the association with the paralysis of one side of vague hemiplegic symptoms of the other; 4th, the supervention of stupidity, associated for the most part with extreme drowsiness, a puzzled enquiring look when awake, a difficulty of getting food down the throat, and cessation of speech—I say a difficulty of getting food out of the mouth rather than paralysis of the mouth and throat, and I say a cessation of speech rather than aphasia or loss of articulating power, because it seemed to me, in watching the cases, that these phenomena were due mainly to stupidity and irresistible tendency to sleep, and not definitely to paralysis or affection of the centres for speech; 5th, the absence of implication of the oculo-motor nerves, and of direct implication of other cerebral nerves; and lastly, death from coma. It is further noteworthy that some of the more striking symptoms of cerebral tumour were either absent or only slightly developed. Headache did not appear to be very severe in any case, and in one is not so much as mentioned; there was practically no sickness; optic neuritis was certainly absent, at any rate in one case, up to within a week or ten days of death; from first to last the patients were free from epileptic attacks, and never had anything that could be termed a fit.

So far as I know from reading, disease of the median portion of the corpus callosum is unattended with marked, certainly with characteristic, symptoms. And I am inclined to think that my cases are in accord with this statement. In all of them the tumour, taking its whole bulk, was very large; and must have taken much longer to grow than the clinical history gave as the duration of the disease. And in all of them the median part of the corpus callosum was evidently the starting-point of the disease, and this body must have been largely involved before the patient complained of definite cerebral symptoms. Indeed I take it that the symptoms the patients presented were due chiefly, if not altogether, to the extension of the disease into the hemispheres, and to the diffused pressure on important parts caused by the great collective bulk of the

tumour, as evidenced by the extreme flattening of the surface of the hemispheres, and the singular absence of both subarachnoid and ventricular fluid. Assuming this explanation to be correct, the progress of a case of the disease I have been describing should be much as follows. First, the occurrence of headache, and other somewhat vague symptoms of progressive cerebral disease. Second, the gradual onset of more or less well-marked hemiplegia. Third, the appearance in a greater or less degree of similar symptoms on the opposite side of the body. Fourth, the coming on of dementia, with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder. And these were, in fact, the chief symptoms which my cases presented, and the order of their sequence. Many other symptoms might of course become superadded in the progress of the case, owing either to accidental circumstances, or to the extension of disease into special parts of the brain. Thus, in the second of my cases, there was distinct aphasia, owing to direct involvement of Broca's convolution; and thus, although sickness and epilepsy were absent in the cases I have adduced, I see no reason why they should be absent from all cases of the kind.

The affections with which, as it seems to me, my cases would be most liable to be confounded are, hemiplegia due to hæmorrhage or embolism, disease of the medulla oblongata and its vicinity, and general paralysis of the insane.

I presume that if the case were observed from its very beginning, it would scarcely be possible to mistake one of embolism or hæmorrhage for one of progressive disease of the corpus callosum. And even if such a case came under observation late and without a history, its later progress would, I think, be sufficiently suggestive to the watchful physician. The second of my cases came to me with a very imperfect history; and, guided by the fact that he suffered from right hemiplegia with aphasia, I certainly assumed for the first few days that he was suffering from cerebral hæmorrhage or softening. But the steady downward progress of the case and the discovery of optic neuritis gave it a different aspect. The difficulty might be increased if the patient, suffering from a recent attack of embolic or hæmorrhagic paralysis, had had

a previous attack of hemiplegia on the opposite side of the body.

Disease of the medulla oblongata and its neighbourhood has so far a resemblance to disease of the corpus callosum, that it is apt to be attended by so-called "cross-paralysis," and by difficulty in chewing, swallowing and speaking. But in this case the cross-paralysis is generally not the result of double hemiplegia, but the consequence of the association of hemiplegia with local paralyses due to direct involvement of one or more of the cerebral nerves. Or to put my meaning in another form: in disease beginning in the corpus callosum, if there be cross or double paralysis, it is owing to the co-existence of more or less complete right and left hemiplegia; whereas in disease of the medulla oblongata, the distinctive symptoms depend on the direct involvement of the nerves of that part, and we are apt to have paralysis not only of the muscles of articulation, mastication and deglutition, but also of the oculo-motor and other nerves.

At first thought, general paralysis of the insane might seem to have no relation to disease of the corpus callosum; and it must be admitted that the differences are, for the most part, much greater than the resemblances. Yet I am bound to confess that my third case seemed to me, when I first saw it, to be not unlike one of general paralysis; and it was only after watching the case for a day or two that I determined that it was not. The patient had incomplete paralysis of all his limbs; he passed his evacuations into the bed; he had (or said he had) difficulty in protruding his tongue; he spoke slowly and indistinctly; he seemed to masticate and swallow with difficulty; his language was flippant in quality; his face was extremely greasy; and he complained of nothing. Further, he had no optic neuritis, and no sickness. Now this combination of symptoms corresponds very fairly to what we meet with in certain cases of general paralysis, at certain stages of their progress. But, on the other hand, there was no inequality of pupils; there was no distinct tremor of the lips in speech; and moreover, his symptoms were those, if of any, of the latest stage of general paralysis, a stage which I suppose could not have been reached in the month or two during which alone the

patient was ill, and certainly could not have been reached unless he had suffered from the epileptiform fits which generally attend the disease.

I have added the fourth case because, although it evidently does not form one of the same group as the other three, it has a close affinity to them, both in its morbid anatomy and in the symptoms which were observed during life. Indeed the symptoms, excepting that they were very much slower in their development, were almost identical with those of the other cases, and might well have justified the diagnosis of tumour starting from the corpus callosum.

The patient was a young woman, whose illness began with headache, giddiness and sickness, about fifteen or sixteen months before her death, which occurred about eight weeks after her admission into the hospital. To the above symptoms, which gradually increased upon her, failure of sight and difficulty in walking (with a tendency to fall backwards) were added in the course of time. On admission, she complained of headache, giddiness, and blindness; she could only walk with assistance, and had a manifest tendency to walk on her heels, and to fall over backwards. She was not sick. She had double optic neuritis. There was no definite paralysis, but the tendon reflexes in the legs were exaggerated. Her symptoms rapidly developed while she remained under observation; she became confined to bed; she passed her evacuations under her; her left arm and leg became manifestly paralysed and somewhat rigid; some weakness of the same side of the face was noticed; her speech became drawling and indistinct; extreme drowsiness came on, and with it a tendency to allow her food to remain indefinitely in the mouth. She died comatose.

The patient complained a good deal of headache, also occasionally had hallucinations, but otherwise was sensible; she was only sick once or twice while she was in the hospital, and she never had a fit. The oculo-motor nerves remained normal.

The post-mortem examination revealed the presence of a very large tumour, springing from the surface of the right corpus striatum and optic thalamus, and the neighbouring parts of the floor of the right lateral ventricle, distending this ventricle and displacing the fornix and septum lucidum (which

were partly incorporated in it) to the left, and involving also the central and anterior part of the corpus callosum which lay over it.

The corpus callosum was much affected in this case, but I cannot attribute any of the patient's symptoms to this cause. The tumour of the ventricle, partly by the pressure which it exerted on the overlying right hemisphere, partly probably by its encroachment to a slight extent on the subjacent corpus striatum, furnishes ample explanation of the clinical phenomena of the case.

I am sorry to have to state, that the exact nature of the tumours in these cases was not (excepting in one instance) determined. I was present, however, at all the post-mortem examinations, and I believe that they were sarcomata.

CASE I.—*Tumour of Corpus Callosum extending mainly into left Centrum Ovale.—Right Hemiplegia.—Loss of memory and intelligence.—Drowsiness.—Coma.—Death.*

C. S., a bricklayer. aged 46, admitted Nov. 13th, 1880.

Has enjoyed good health up to ten weeks ago, when he began to suffer from severe frontal headache, and was compelled in consequence to give up work. He had no vomiting or other symptoms. He continued in this state up to three or four weeks before admission, when his right hand became weak, his memory began to fail, and his mind to be confused. It is said too that his sight became defective. His headache at the same time diminished. In the course of a week or so, the weakness of the right hand and arm had increased, the leg had become implicated, and his loss of intelligence had become more pronounced. About a fortnight since, he took to his bed, and at that time he fancied he was not in his own house. He has gradually been losing the power of walking and even of standing without assistance. He has been very drowsy in the daytime, but wakeful at night. He has felt nausea, but not been sick; and his appetite has been fair. He has retained power over his rectum and bladder.

He is a fairly well-nourished man; but his friends say that he has lost flesh. Cannot stand; but when supported in walking

drags his right leg. He does not answer questions readily; will not do what he is told to do; and does not seem to understand all that is said to him; slow in thought and movement. Complains of pain in left frontal region. Face apparently a little smoother on right than on left side; but he opens and closes his eyes equally well; and the mouth is not drawn to left. Tongue protruded straight. No squinting; no double vision; says that he sees well; pupils equal; hearing good. Right arm and leg considerably weaker than left; no impairment of sensation. Patellar-tendon reflex increased in right leg. No affection of thoracic or abdominal organs. Tongue a little coated. Urine free from albumen. No loss of control over emunctories.

On the 18th he was very drowsy, lying with eyes half-closed, and refusing or unable to speak. The day before he was tested in reference to his knowledge of the names of things. He named objects fairly readily; and the only mistake he made was to call an inkstand a penholder. It was noticed that the left eye closed less quickly than the right. About this time he began to pass his evacuations into the bed.

He remained in much the same condition. And on the 28th it was noted that he was still drowsy, and could only be got to speak by repeating questions loudly two or three times; that he ate very slowly; and that he allowed food to remain in his mouth for five minutes or more without attempting to swallow it.

On December 3rd it was observed that the weakness of the right arm and leg had increased: and that the left eyelid did not close so perfectly as the right. He remained in the same drowsy state.

He gradually became weaker and more drowsy; and passed into a condition of complete coma, in which he died on the evening of December 8th. His temperature had been normal during the greater part of his stay in the hospital. But it rose to 100·6 on the evening of the 7th, and on the day of his death varied between 101·2 and 104·8. An hour before death it was 103·6.

Post-mortem examination.—All organs were free from disease except the brain. The membranes were healthy; and there

was no excess of fluid on the surface, or in the ventricles. The vessels at the base also were healthy. A soft, spongy, vascular, reddish-brown tumour was found occupying the corpus callosum in the greater part of its extent, both from before backwards, and from side to side. It was largest anteriorly, where it occupied the anterior genu; and it extended thence on either side into the centrum ovale, chiefly however on the left side. On the right side it ceased opposite the extremity of the caudate nucleus: but on the left it extended to the expansion of the internal capsule. Neither the convolutions, nor the optic thalami, nor the corpora striata were involved. But the tumour adhered slightly to the upper surfaces of the anterior extremity of the left caudate nucleus.

CASE II.—*Tumour of anterior half of Corpus Callosum and each hemisphere, and on the left side involving the frontal convolutions.—Right Hemiplegia.—Aphasia.—Optic Neuritis.—Rhythmical Tremors.—Stupidity.—Death, with high temperature.*

Joseph McK., a sailor aged 41, was admitted under my care on the 19th of April, 1882.

Four weeks ago, while at sea, his speech became affected, and his manner dull. About the same time, or a little later, the right arm and leg got weak. He had no fit. The symptoms have increased upon him; and for two or three weeks he has kept his bed. He has not been emotional; and has retained power over his evacuations.

There is complete right hemiplegia. He cannot move either arm or leg; the mouth is drawn to the left; and the tongue is protruded towards the right. The leg is somewhat rigid, but the arm is limp. The tendon and superficial reflexes are normal on both sides; and there does not appear to be any impairment of sensation. No affection of ocular muscles; pupils contracted and equal; sight apparently good. No difficulty in deglutition. He can say a few words, and seems to understand fully what is said to him; but is evidently aphasic to an extreme degree. Does not repeat words to dictation. Hearing imperfect, especially on right side.

Tongue clean; no sickness; bowels open. Heart healthy. Urine free from albumen, sp. gr. 1025. Temperature normal.

At this time it was believed that the patient was suffering from an ordinary attack of hemiplegia due to hæmorrhage; but on the 24th his eyes were examined with the ophthalmoscope. It was then seen that the right optic disc was hazy and woolly, that some of the vessels had white lines along them, and that the veins were slightly dilated; but that the left eye was healthy.

27*th*.—He is in much the same condition, excepting that his loss of speech has become more marked. He now only says "no" in answer to every question. But, though there is doubtless some impairment of intelligence, he seems to understand all that is said to him, and tries to reply and do what he is told. He evidently recognises all objects that are shown him, and even their names when repeated to him. He answers "no," but at the same time shakes his head in negation, or nods affirmatively, as the case may be. He can copy letters imperfectly with his left hand, but cannot write. He does not recognise printed words or letters; at any rate he recognises them very imperfectly. He has some headache on the left side. The paralysis remains as before; but it is thought that there is also some impairment of sensation on the paralysed side.

A further examination with the ophthalmoscope was made by Mr. Nettleship. He confirms the description already given of the right disc; but he observes that the left disc is not absolutely clear; that at least three of the vessels bend abruptly just outside its margin; that there is slight neuritis.

From this time the change in the patient from day to day was slight; but on the whole he slowly got worse. He suffered more or less from headache, always referred to the left side. The paralysis of his limbs remained complete; but the arm after two or three weeks grew rigid; and the tendon and plantar reflexes in the right leg became exaggerated. No squint appeared, but the left pupil became at first smaller and subsequently larger than the right. A difficulty in protruding his tongue came on, and saliva at times dribbled from his mouth, but he could masticate and swallow. The slight

hemianæsthesia continued. He lost the power of speech absolutely, and gradually became dull and stupid; and, though looking at and watching intently any one who addressed him, apparently made no effort to respond. Occasionally he seemed fretful; but on the whole he was utterly apathetic. The pain in his head was relieved once or twice by the application of a few leeches to the temples.

On the 20th of May the following note was taken:—The patient lies in an apathetic condition, taking very little notice of anything, and seeming not to understand when spoken to. His chief employment is gnawing a flannel roller or his handkerchief. The paralysis remains complete; and the arm, but not the leg, is rigid. He seems unable to put out his tongue, and is wholly speechless. But he can swallow, and has a good appetite. The pupils are equally dilated, but from the effects of atropine. He apparently has pain on the right side of the head. His evacuations are passed unconsciously. For the first time his temperature rose to-day to 100·8.

From this day he gradually sank. He got more and more dull and stupid. Rhythmical tremors came on in his arms, but only when they were moved voluntarily or by force. The pupils became unequal, the right being the smaller. Profuse perspirations broke out, and the temperature rose. On the 26th it reached 102; on the 27th, 103; on the 28th, 105·4; and just before his death (which occurred a little after 8 P.M. on the 29th), 107·4.

Post-mortem examination.—Dura mater normal. There was no excess of fluid either in the subarachnoid tissue or in the ventricles, and no trace of meningitis. The surface of the left cerebral hemisphere was perhaps a little more prominent than that of its fellow. On making careful transverse and longitudinal sections of the brain, a large infiltrating ill-defined growth was found, involving the anterior half of the corpus callosum; and extending thence, on the left side into the substance of the hemisphere, so as to occupy nearly the whole of the anterior half of its white matter, together with the grey matter of the second and third frontal convolutions and the ascending frontal; and on the right side so as to implicate a considerable portion of the right centrum ovale in the corresponding

situation, but not reaching the grey matter. The tumour was above the level of the corpora striata and optic thalami, and did not affect them, or the internal capsules. The new growth was greyish in tint, and looked soft. But it was really tough, and cut crisply. The portion occupying the left hemisphere was spongy; that on the right side was more homogeneous in texture, but presented a series of concentric markings.

All the other organs were healthy.

CASE III.—*Tumour of anterior three-fourths of Corpus Callosum, extending into the white matter of the hemispheres. —Imperfect paralysis, mainly of the left side; difficulty of speech; great drowsiness and stupidity.—Death.*

J. H. P., an accountant, 51 years of age, was admitted under my care on the 21st of July, 1884.

He had been a strong and healthy man until ten weeks previously, when he was attacked with quinsy. He was ill with this complaint for a fortnight or more, and remained feeble for a few weeks longer. But he finally got quite well; and is said to have remained well until ten days before admission. At that time it was supposed that he was overcome by the heat of the weather; at any rate he became weak, and his weakness has increased from that time. He was sent to the hospital with the statement, that he was suffering from diphtheritic paralysis.

A well-nourished man. He was very drowsy, but when fully awake appeared to be quite sensible, and, though speaking slowly, made pertinent, indeed somewhat flippant, answers to questions. But his articulation was indistinct. He did not fully open his mouth or put out his tongue when told to do so, and said that he could not. The right side of the face was thought to be a little less mobile than the left. There was loss of power in both upper extremities; and the left was flexed and somewhat rigid. The legs also were weak. The right he seemed unable to move, the left he moved slightly. The tendon reflexes were present and normal; as also were the cutaneous reflexes. There was no loss of sensation anywhere. There was no nystagmus; no squint. The pupils were of

normal size, equal and acted to light. His urine was passed into the bed.

No affection of heart or lungs. Pulse 80. Abdominal organs all healthy.

I saw him the next day, and his condition accorded in the main with the above description. But his drowsiness had increased; and it was very difficult to wake him or to keep him awake. He seemed to have a little difficulty in swallowing, and in getting the food out of his mouth. This, however, might have been due to his sleepiness. He answered questions only when fully roused, and then sensibly, though slowly and somewhat indistinctly. There did not appear to be any actual aphasia. He said that he had little or no pain. The eyes were quite normal as to pupils and movement; and ophthalmoscopic examination revealed nothing amiss. The evacuations were passed unconsciously; and the urine, which had a specific gravity of 1032, was acid, and free from albumen and sugar.

He continued in much the same state for the next two or three days. On the 26th I made the following note:—"Patient still passes everything into the bed. He is much less drowsy than when I last saw him. Hears, but does not seem to understand what is said to him. When spoken to loudly, makes some attempt to do what he is told or to answer, but his speech is mumbling and unintelligible. He keeps his eyes intently fixed on my face, and follows me with them when I move. He has a little difficulty in chewing and swallowing, and occasionally chokes. There is no paralysis of ocular muscles, or affection of the pupils. The left side of the face is perhaps a little weaker than the right. Moves right arm more than left, but both are weak and somewhat rigid. Legs as before. Sensation perfect. Face very greasy."

Henceforth his symptoms underwent scarcely any change. He became perhaps more drowsy and difficult to wake, and difficult to feed on account of his drowsiness. There was no obvious increase of paralysis; but certainly no improvement in this respect; and his evacuations were still discharged into the bed. He occasionally tried to speak; but the sounds he uttered were unintelligible. On the 28th his pulse was 112,

but his breathing normal. On the 29th his urine, which was still free from sugar and albumen, was strongly alkaline, and its specific gravity 1035. On the evening of the 30th he had an attack of dyspnœa, attended with rattling in the throat; and the respirations rose to 54 in the minute. The dyspnœa subsided in the course of a few hours. During the whole of the 31st he was unconscious. It was noticed that both arms, but chiefly the left, were rigid. At six in the evening he began to breathe stertorously, and he died three hours later.

It is noticeable that the patient never complained of, or seemed to suffer from, headache; that there was never any sickness; and that he had never had anything of the nature of a fit. His temperature, during the first three days, varied between 98·4 and 99·8. During the next seven days, it rose every evening to between 100 and 100·6. Shortly before death it attained 102·2.

Autopsy.—On removing the dura mater the convolutions were found universally flattened, the surface of the brain dry, and the pia mater and veins everywhere remarkably congested. But there was no appearance of inflammatory effusion and there were no adhesions. No fluid was found at the base of the skull; and none escaped from the lateral ventricles as the brain was removed. The arteries were all healthy. On separating the hemispheres, the corpus callosum was seen to be much arched from before backwards; and the surface of its anterior two-thirds, as far outwards on either side as it could be traced, was yellowish and irregularly modulated. On further examination, it was found that the anterior three-fourths of the corpus callosum, and the corresponding portion of the fornix (including the anterior genu of the former, and the anterior pillars of the latter) were the seat of a growth, which had caused the affected part of the corpus callosum to be at least as thick again as natural, and the fornix to measure in some places about half an inch in thickness. The general form of these bodies was, however, retained. The diseased fornix was grey, translucent, soft and vascular, and had the appearance of being the seat of growing sarcoma. The corpus callosum presented patches of the same kind of material, but was mainly the seat of a toughish opaque buff-coloured

reticulum with coarse irregular meshes, occupied in part by the growth above described, in part by a gelatinous kind of material, or a serous fluid. The diseased textures extended from the corpus callosum on either side into the anterior half of the centrum ovale majus, which was involved in almost its whole extent above the level of the corpus striatum. The disease was somewhat more extensive and more advanced on the right than on the left side; and the affected tissues had exactly the same characters as those presented by the central part of the corpus callosum. There was no very definite limit between the tumours and the brain-tissue around; which latter was soft and much congested. Microscopic examination showed that the tumour was a sarcoma, undergoing in places degenerative changes. The grey matter of the convolutions was everywhere healthy; the ganglia at the base were also altogether healthy; and the lateral ventricles contained only a minute quantity of fluid. There was no disease elsewhere in the brain; or in the body.

CASE IV.—*Tumour springing from floor of right cerebral ventricle, and involving the septum lucidum, fornix, and corpus callosum.—Headache, optic neuritis, blindness, left hemiplegia, drowsiness, coma, death.*

Florence M., an unmarried woman 25 years of age, was admitted into St. Thomas's under my care on the 13th of June, 1883.

She is said to have been a delicate woman, but to have suffered from no serious illness until fourteen months ago, when she had a sharp attack of small-pox. She dates her present illness from that time. The first symptoms were pain at the top of the head and across the eyes, giddiness, sickness, mainly in the morning, and weakness in the legs. These symptoms gradually became more severe; and latterly her eyesight has been failing. For some time past, when standing, she has had a tendency to fall backwards. She has been quite sensible all along, and has had no fits. No history or sign of syphilis.

She is a florid, healthy-looking woman, perfectly sensible,

and ready of speech. She complains of vertex headache, of blindness, and of difficulty in walking. The headache varies, but is never severe. The eyes are very prominent, but otherwise look natural. Their movements are consensual, and normal, excepting that the corneæ are never turned completely into the outer or inner canthi. The pupils are equal, but act neither to light nor to accommodation. Sight is almost gone; the patient can only distinguish light from darkness. There is double optic neuritis; the discs are much swollen, but little congested, the vessels are tortuous and slightly obscured at the discs. There is no paralysis of either side of face or tongue, and mastication and deglutition are unaffected. Sensation is perfect in head and face. Hearing and taste are normal. Says she has lost smell, but she recognises ammonia and acetic acid. There is no paralysis, rigidity, tremor or wasting of arms or legs. But the legs present exaggeration of plantar and tendon-reflexes, with clonus at ankles and knees. There is no apparent impairment of sensation. But she says she feels as if walking on wool. She can stand and walk, though with difficulty. The gait is a little peculiar. There is no ataxia, but she walks very much on her heels, and has a constant tendency to fall backwards with an inclination to the right. There is no affection of bladder or rectum, and the catamenia are regular. No bed-sores. Thoracic and abdominal organs apparently all healthy. Urine free from albumen.

The patient rapidly got worse after admission. In three or four days it was found that she could not stand without being supported on both sides. On the 18th it was noticed that she was excited; and on that day and for several days subsequently she had optical hallucinations, addressed imaginary persons, and said she saw soldiers all dressed in blue. This condition disappeared after a few days, and apparently did not recur. Then and thenceforth she was emotional, sometimes low-spirited, but more frequently jovial. On the 23rd and 24th, she passed her evacuations unconsciously, and for the most part they escaped without control during the remainder of her life.

Early in July she began to suffer from drowsiness. About the 8th it was observed that the mouth was drawn slightly

to the right when she laughed or spoke; that the right eye closed a little more than the other when she smiled; and that her speech was slow and drawling.

On the 15th, it was remarked that the right arm seemed to be weaker than it had been: and that the left was occasionally rigid.

Subsequent observation confirmed the fact of slight left facial palsy; and that of comparative weakness with rigidity of the left arm and leg, the rigidity being occasional. Once or twice the tongue was protruded towards the left.

Her headache continued, and on two or three occasions she was sick. From the time of its commencement, her drowsiness, with occasional intermissions, continued, and increased upon her. And for the last week or two of her life it was so constant and so profound that there was the greatest difficulty in rousing her even for a moment; and it became a long and weary work to give her nourishment, inasmuch as she went to sleep with the food in her mouth, and if left, would allow the food to remain there for half an hour or more at a time. She generally lay with the tongue lolling out of her mouth.

Finally her drowsiness passed into coma, and in this condition she died early in the morning of the 5th of August.

She never had a fit. Her temperature throughout her illness was generally normal. Occasionally, however, it rose to 100, and shortly before death reached 100·8.

Autopsy.—A well-nourished young woman, with very crooked legs, the consequence of rickets.

On opening the head, the convolutions were found to be flattened, the surface generally dry, and the pia-mater uniformly congested. On removing the brain, there was no accumulation of fluid at the base, and no visible sign of inflammation; but it was noticed that the space between the crura cerebri was widened, and that the optic tracts were stretched. The infundibulum, however, was not prominent, and no fluid oozed from this part. On slicing away the upper part of the hemispheres, the corpus callosum was found to be unusually arched from before backwards, and also from side to side, as though raised by some subjacent accumulation, situated mainly towards its anterior part. The dissection was now

conducted with care, and the following are the results which were arrived at:—A very soft, pinkish, vascular growth, much lobulated on the surface, measuring about 4½ inches from before backwards, 3½ from side to side, and from 1 to 2½ in thickness, sprung mushroom-like from the surface of the right corpus striatum and anterior part of the right optic thalamus, and from the floor of the anterior cornu and of the ventricle external to the ganglia. The subjacent brain-substance was scarcely, if at all, encroached on by it. The growth occupied the whole of the distended right ventricle, excepting its descending and posterior cornua; and, pushing the septum lucidum over to the left side, stretching it, and incorporating it to a large extent, formed a lobulated protrusion into the left ventricle. The fornix also was largely displaced to the left and stretched over the tumour; and the greater part of it, like the septum, was incorporated in it and lost. The corpus callosum, in its central area, was stretched over the tumour, adherent to it, and partly destroyed by it. Although the tumour had obviously originated in the right ventricle, about a fourth of the bulk was to the left of the mesial line. The third ventricle was a little displaced, but normal in all other respects. There was very little fluid in the cavities. The brain, with the exceptions above noted, was quite healthy. The tumour, judging from hasty examination, was a sarcoma.

Abdominal and thoracic viscera all healthy.

MUSCULAR ATROPHY, AFTER MEASLES, IN THREE MEMBERS OF A FAMILY.

BY J. A. ORMEROD, M.D.

THE two children whose cases form the principal part of this paper were under observation, as out-patients at the National Hospital for the Paralysed and Epileptic, from February to September 1883. In August 1884, I heard from their mother that they were still in the same condition. They had been, at an earlier stage, under the care of Dr. Barlow, who had noted the paralysis as peculiar, and who has kindly seen them for me again.

CASE I.—Annie W., æt. 17, was brought by her mother to me on January 30, 1883. Her legs were crippled, though she was able to walk; both hands were partially paralysed. The history given was this: she had measles between 6 and 7 years of age; about a month after she recovered from them, one foot (the right ?) began to "turn," the toes, according to the mother, pointing downwards and inwards. Tenotomy was performed at the Orthopædic Hospital. After this the other foot began to turn in the same way. About seven years ago (*i.e.* three or four years after the original attack of measles) tenotomy was again performed at University College. This affection of the legs did not come on suddenly, but took about a month to develop.

More recently the hands have become weak. This began five years ago. The mother first noticed that the girl used her hands awkwardly and stuck her knuckles out. The hands have got gradually worse, especially the thumbs, during the last two months.

She is subject to headache; also to shortness of breath and wheezing at the chest. Her menses are regular and natural, but preceded by much pain across the back and lower abdo-

men. With these exceptions her general health is perfectly good.

Her condition while under observation was as follows. Her legs were much wasted below the knees; the muscles of the thighs were not so well nourished as they should be. In cold weather there was much disturbance of the cutaneous blood-supply in the legs; sometimes the calves were blue and cold, with bright red patches on them like chilblains going to break; sometimes, in addition to the coldness and congestion, the whole leg below the knee was so swollen as quite to hide the wasting of the muscles. The tendon-reactions were present at both knees. The induction current produced no action when applied to the muscles below the knees, to the peroneal nerves—to the vasti—to the muscles at the back of the thighs (the last doubtful). The constant current also produced no action when applied to the peroneal nerves, or (so far as could be made out) to the muscles below the knees.

As to the condition of the hands and arms, for which she principally sought advice—there was well-marked wasting of the balls of both thumbs and of the intrinsic muscles of the hands generally; and wasting, though less apparent, of the muscles on the forearms, both extensor and flexor aspect. The fingers were in a position of semi-flexion at their two distal joints; their metacarpo-phalangeal joints being almost straight. All voluntary movements at the elbow- and shoulder-joints were perfectly retained; but extension of the wrist and fingers was possible, on the right side, only to a very slight degree; on the left side, not at all. Flexion of the wrists and fingers was possible, but to a slight degree only, on both sides. The left hand was said to be the worst.

Electrical examination, on two or three occasions, (it should be mentioned that she could not bear very strong currents) gave the following results:

(*a*) The induction current produced no action in the muscles of the thumb-ball or intrinsic muscles of the hands, nor in the muscles of the forearms; on the other hand, the muscles of the upper arms, and the deltoids, rhomboids, &c., acted normally.

(*β*) Reaction to constant current.

In muscles of thumb-balls present but diminished[1] [on L. side AOZ = KSZ: on R. side ASZ = KSZ]: later, no reaction could be obtained.

In extensor muscles of forearms: on L. side, absent: on R. side diminished, without qualitative change: later, no reaction on either side.

In flexors: on L. side absent; on R. side, diminished without qualitative change. But the flexor longus pollicis and pronator radii teres seemed to act normally.

In ulnar nerve at elbow, and median nerve at wrist, no reaction.

To summarize; the affected muscles showed absence of faradocontractility, and absence or diminution of galvano-contractility, with qualitative change in one instance only.

From Jan. 30th to May she took codliver-oil and syrup of phosphate of iron; from May to September, hypophosphite of soda. Meanwhile she had the constant current to the upper limbs. Complaint was made in July of incontinence of urine, not at night, but while she was walking about. This had been noticed once before; but it did not appear to trouble her much. In the condition of her limbs there was no change; and her attendances became so irregular that she had to be discharged.

Case II.—Alfred W., æt. 9, brother of last patient, was first brought on Feb. 20, 1884, the history being that four years ago, after measles, weakness of both legs set in. This came on gradually, in about three months' time. But during the last few weeks, his mother had thought that the hands were getting weak.

The muscles of the lower limbs were much wasted below the knees, above they were apparently healthy; the patellar tendon reactions were exaggerated on both sides. The right thumb-ball was flattened as compared with the left; the induction current acted on *neither* thumb-ball. He appeared slightly feverish, and after this visit was laid up at home with bronchitis, so that I did not see him again till April. He had then

[1] *I.e.* required a considerably stronger current than the corresponding muscles in my own person. The estimation was made with a galvanometer, to avoid fallacies due to inaction of cells and variations of cutaneous resistance.

got thinner, and his mother thought that his hands were worse.

The following notes were taken between April and September.

There is wasting of both lower limbs, worst on the right side; but only below the knees. There is no vaso-motor disturbance, as in his sister's case. He wears orthopædic boots; when he walks without them, the right foot turns in. He can flex and extend the knee-joints freely; the patellar tendon-reactions are lively on both sides. As to the upper limbs, there is now evident wasting of the muscles of *both* thumb-balls, but still most marked on the right side. He cannot move the right thumb so freely as the left. The fingers tend to assume a position of flexion at their two distal joints, and extension at their metacarpo-phalangeal joints. This is particularly noticeable while he is using the hands. It is exactly the pose[1] which the mother noticed in an early stage of his sister's disease. He can, however, flex and extend the fingers at will. The forearms and upper arms are unaffected; the tendon reactions at wrists and elbows are lively.

Systematic electrical examination was impossible, on account of the child's nervousness, but the following facts were made out.

(*a*) A fairly strong induction current (which acted readily on my own thumb-muscles) had no action on his thumb-muscles, nor on the first dorsal interossei (the latter doubtful), but acted on the muscles of the forearms, both extensor and flexor aspect. It did not act on the muscles below the knees, either anterior or exterior group, nor yet on the vasti, though these latter did not appear wasted.

(*β*) A moderate constant current acted on the muscles of the right[2] forearm, extensor aspect, KSZ > ASZ; but had no action on the thumb balls.

The constant current did not act on the muscles of the legs, but acted on some at least of the thigh muscles.

The electrical conditions then, so far as they could be made

[1] Due to paresis of the interossei.

[2] The left forearm, and the other muscles of the right, were not examined, but may perhaps, from their normal appearance and faradic reactions, be presumed normal also.

out, were the same as in his sister's case; viz. absence or much diminution of contractility, in the affected muscles, to *both* currents.

He was treated with faradisation to his hands and forearms, and with the same medicines as his sister. He did not improve; indeed the peculiar pose of his hands became, if anything, more marked.

In neither of these cases was any complaint made of sensory disturbance. There were no fibrillary twitchings, and no rigidity. The affected muscles were, as I have said, wasted or wasting; and I did not notice enlargement of any other muscles; though I admit that I did not specially look for it. Being told by the mother that the father had been paralysed in one leg from infancy, I visited him at his cottage, and made the following notes.

Case III.—William W., æt. 56, cab-cleaner, healthy in appearance, though grizzled and looking older than his years. Ever since he can remember, his right leg has been in its present condition. He was told by an elder sister[1] that he had measles very badly when 12 months old, and that the right foot began to turn when he was about three years old. His family seem to have connected the affection of the leg with the measles.

There is a decided difference in girth between the two lower limbs, especially below the knee, as follows:

Largest girth of left calf, 12·5 inches .. of right, 9·5 inches,
„ „ „ thigh, 16 „ .. „ „ 15 „

In length, no difference between the two. Patellar tendon reactions well marked on both sides, but the left greater than the right. The muscles below the right knee are extremely feeble; the foot hangs quite limp at the ankle, he cannot point the toes at all; he says he always walks on the heel; and he can only evert the foot slightly.

He cannot extend the right knee-joint with the same facility as the left.

[1] This was told him before the birth of his two children, now paralysed. I mention this to show, that the sequence of events in his case was not made to suit theirs.

The left leg is perfectly natural, the arms and hands are natural, and he is positive that he never had anything the matter with them. I had no opportunity of making an electrical examination.

Evidently his condition may have been due to an old attack of infantile paralysis, but I do not feel certain that it was so. The history, such as it was, pointed to a gradual onset; and the bones had grown to the same length as those of the other limb. In former years a doctor has taken much interest in his condition, had him photographed, &c.

Of five other children (besides my two patients), one was smothered in bed, four are alive and healthy. Two of these are grown up. All had measles at one time or another. The mother was one of a remarkably large family of children, and she knows of no nervous complaint among them. The father (Case III.) was one of four, all healthy; of these a sister committed suicide at 15, but he is confident she was provoked to it, and was not insane. His father, mother, and paternal grandfather, were all healthy.

The points that deserve notice are

(1.) The connection with measles.

(2.) The youth of the patients.

(3.) The fact that brother, sister, and father were paralysed.

(4.) The possible nature of the disease.

The measles were not apparently severe in the children's case. No doctor was called in; but from the description the mother gave me of the coryza and the rash, and from the fact that measles was known to be in the neighbourhood, I have little doubt that this disease was, as she said, measles. The two children who became paralysed did not have them at the same time; one of the others had them at the same time as Alfred, but did not become paralysed; neither did any of the neighbours' children, so far as was known to the mother. There was therefore nothing in the nature of the epidemic specially tending to cause paralysis. On the other hand, considering the uncommon nature of the paralysis, it seems unlikely that the connection with measles should have been in the case of both children (and in that of their father?) a mere chance sequence. The exciting cause was probably the

measles; the predisposing cause, a family proclivity to this form of paralysis.

This brings us to the other points, which it will be convenient to consider together. What was the nature of this paralysis? The three clinical types which suggest themselves are: (1) acute anterior poliomyelitis or ordinary infantile paralysis; (2) chronic[1] anterior poliomyelitis or paralysie spinale antérieure subaigue (Duchenne); (3) progressive muscular atrophy.[2] We may dismiss the first of these, with which the onset and course of the disease entirely disagree. As to the second, chronic anterior poliomyelitis, the youth of the two patients might have been held, till recently, to justify us in dismissing this also. But cases recently published[3] by Professor Erb and Dr. Hughes Bennett show that chronic anterior poliomyelitis does occur in children, however rarely. Indeed, between Dr. Bennett's first case and my own there are some close similarities. Dr. Bennett's patient suffered from whooping-cough and *measles;* a month afterwards, slight weakness and unsteadiness of the gait was noticed; still later, awkwardness of the hands; there was cyanosis of the legs, and diminished response both to faradism and galvanism. There remains, however, this difference, that in my patients there has been no attempt at recovery, though the disease is now of five and ten years' standing; whereas both Erb and Bennett emphasise the fact that their patients recovered, as being confirmatory of the diagnosis.

Moreover if it is rare for chronic anterior poliomyelitis to occur in children, it is, so far as I am aware, unknown that it should run in families. This is not the case with progressive muscular atrophy; which, as is well known, frequently attacks many members of a family. These also are just the instances in which the disease begins in early life. For recent information on the subject of "hereditary" muscular atrophy, I may refer the reader to an article by Zimmerlin, recently abstracted by Dr. Beevor.[4] As regards the progressive muscular atrophy

[1] Better perhaps called subacute, to avoid confusion with (3).

[2] This last probably comprehends several forms.

[3] 'Brain,' Nos. 21 & 23, "On Chronic Atrophic Spinal Paralysis in Children."

[4] Zimmerlin, 'Zeitschrift für Klin. Med.,' Band I., Heft I. 'Brain,' No. 26, p. 285.

of youth, Erb recognises this as a distinct type and assigns to it the following characteristics.[1] It begins insidiously in childhood or youth, usually in the shoulder or arm muscles, sometimes in the legs or back. The pectorals trapezii latissimi rhomboids and long muscles of the back are at a certain stage almost always involved; biceps, brachialis, and supinator longus very frequently; and the triceps later. Among other muscles, the deltoids and *small muscles of the hands are always spared*, and the muscles of the forearms for a long time. In the lower extremity, the glutæi, quadriceps, peroneals and tibialis anticus are usually attacked. The districts of the bulbar and cerebral nerves are not invaded.[2] While some muscles are soft and flabby, others may remain firm. There are no fibrillary twitchings. The irritability of the muscle to mechanical stimuli disappears. The patellar tendon reflexes are preserved. Distinct reaction of degeneration has never been demonstrated to exist.[3] The course of the disease is very chronic. This form of disease, Erb suggests, when it attacks several members of one family, constitutes the "hereditary" or "family" form of progressive muscular atrophy; and when it is complicated with formation of fat in lieu of the atrophied muscle, constitutes pseudo-hypertrophic muscular paralysis, which is also, as is well known, a "family" disease. He thinks that the disease begins in the muscles themselves, not in the cord or nerves.[4]

Now as regards the present cases, the age of the patients, the family predisposition, the absence of distinct reaction of degeneration,[5] the preservation of the tendon reactions—all suggest that they should be classed as instances of this

[1] The original article will be found in 'Deutsch. Archiv für Klin. Med.,' Band 34, s. 467. I quote from an abstract by Strumpell, 'Neurol. g. Centralblatt,' 1884, p. 200.

[2] Duchenne states that progressive muscular atrophy in children begins in the face. E. Remak ('Neurolog. Centralblatt,' 1884, p. 337) gives a case of Erb's juvenile type, with complete facial diplegia.

[3] There is a simple diminution of contractility to both currents ('Neurolog. Centralblatt,' 1883, p. 453).

[4] Recent pathological observations by Landouzy and Dejerine confirm this. See 'Lancet,' Jan. 26, 1884.

[5] Qualitative change was found once only the galvano-contractility was diminished or absent; see above.

juvenile form of progressive muscular atrophy. But the order of invasion, at least as regards the upper limbs (first muscles of hands, then of forearms), certainly does not agree with this view.

I am compelled therefore to leave the exact nature of the disease undecided; perhaps the further course of the cases may decide it; but as it seems unlikely that I shall have further opportunities of investigating them, I venture to record them as they at present stand.

LESIONS OF THE NERVOUS SYSTEM ETIOLOGICALLY RELATED TO CUTANEOUS DISEASE.

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THE following observations are founded chiefly upon the following works:—'Die Neuropathischen Dermatonosen,' by Dr. Ernst Schwimmer, of Buda-Pest; 'Recherches Cliniques et Anatomo-pathologiques sur les Affections Cutanées d'Origine Nerveuse,' by Dr. H. C. Leloir, one of the Paris Theses; 'des Lésions Trophiques consécutives aux Maladies du Système Nerveux,' by Dr. X. Arnozan, another Thesis; 'Nature de l'Influence de l'Innervation sur la Nutrition des Tissus,' by Dr. E. Lahousse,'—a prize essay of the Royal Academy of Medicine of Belgium; 'Injuries of Nerves,' by Weir Mitchell; 'Maladie du Système Nerveux,' by Charcot; 'Diseases of the Spinal Cord,' by Erb, in Ziemssen, 'Trophoneurosen,' by Samuel, in Eulenburg's 'Encyclopædia,' &c.

Schwimmer and Leloir occupy themselves chiefly with the view of proving that certain diseases of the skin are dependent more or less directly upon the nervous system. Leloir's is an essay setting forth observations, mainly original, on the changes in the cutaneous nerves in certain diseases of the skin, which we shall consider presently, together with a review of the clinical facts which support the nervous origin of these diseases. Schwimmer's is a more comprehensive treatise, and is the most recent; and while in his zeal he has included some skin affections whose nervous origin will scarcely be accepted by most authorities, his careful and elaborate work most ably sets forth all the facts, cases and arguments that can be adduced in favour of this view, and he has brought together in an accessible form a large number of valuable cases that

were scattered over the immense range of medical literature, and has also contributed many important cases from his own practice. Future workers in this field will therefore find their labour much simplified.

Schwimmer and Leloir have considered the subject from the skin side; I propose to take it up from the nervous system end, chiefly, but not exclusively, limiting myself to cases where definite lesions of the nervous system have been anatomically established.

While there are a large number of cases upon record in which lesions of the cerebrum have been associated with cutaneous eruptions, it is rare to be able to exclude the possible influence of the cord or other parts of the nervous system; moreover there are not a few instances in which the brain has evidently played an important part in the production of the eruption, and yet no anatomical change is likely to be present. Such are cases of disease of the skin due to mental emotion, as in alteration in the distribution of pigment, e.g. sudden blanching of the hair from fear, anxiety, or grief; though these are well known and authenticated, a case occurring under medical supervision is of interest. Eulenburg[1] and Landois had a patient in the hospital, in whom greyness of the hair and beard appeared in the night from excessive terror, while suffering from delirium tremens. Many instances of pigmentation changes, the result of mental emotion in other parts of the body, are on record. The frequency of vitiligo in the insane, and among epileptics, might be cited as evidence that the brain exercises an important influence upon pigmentation, both as to increase and decrease, doubtless through the sympathetic centre; but this influence is too vague for our present purpose. More to the point, but still of uncertain interpretation, is Bourneville and Poirier's[2] case of partial discoloration of the skin, in association with a tumour of the fronto-parietal lobe of the left side. Eruptions are frequent in general paralytics, especially pemphigoid bullæ. Déjérine records one where, twelve days before death, pemphigus broke out on the extremities, and, post-mortem, the nerves underneath

[1] 'Archiv für Path.-Anat.' Bd. xxxv. p. 575.

[2] 'Progrès Médical,' 1879. Quoted by Leloir, p. 38.

the bullæ were found degenerated; there was also sclerosis of the lateral columns, as well as diffuse peri-encephalitis. It is impossible here to lay all the blame on the encephalitis, on the one hand, nor on the peripheral nerves, as Leloir appears inclined to do, and all analogy resists the assumption that the lateral columns were responsible. Indeed, if the observer were not so careful and skilful as Déjérine, one would suspect that some degeneration of the posterior columns had been overlooked. This case is cited as an example of how complicated the problem is as regards the cerebrum. Moreover, that these eruptions are generally due to the cord rather than the brain, is suggested by the fact that the bullæ, so Dr. Savage tells me, are always on the lower extremities in general paralytics, while the frequency of posterior sclerosis in them is well known. A somewhat more definite connection existed in a general paralytic I saw at Bethlehem, with Dr. Savage, in whom melancholia alternated with excitement. In the melancholic state his skin was cold, clammy, and intensely livid, but became normal during exaltation. Even with regard to vitiligo the cerebrum shares only an influence with other parts; thus, Bulkley,[1] confirmed by Debove and Barthélémy, speaks of the frequency of vitiligo in ataxics; and Leloir gives cases of peripheral nerve-changes in vitiligo patches when the presumption was against cord disease, though a negative was not anatomically proved.[2] But the loss of brain influence may affect the distribution of an eruption. Thus Janin de Saint-Just[3] records a case of a hemiplegic in whom scarlatina supervened; for two days the eruption was limited to the healthy side, and when it did attack the paralysed side, was pale and fugacious.

Chevalier,[4] on the other hand, saw a case of small-pox, in which the eruption was confluent and hæmorrhagic on the incompletely paralysed side, and discrete and normal on the

[1] 'Archives of Dermatology,' 1878.

[2] Leloir, loc. cit. p. 33, &c.

[3] Quoted by Sané, Dict. Encyclop., tome vii. Series III. p. 307 — article "Scarlatine."

[4] 'Développement des Erupt. Cutan. chez les Neuropathies.' Thèse de Paris, 1878.

healthy side. Bouilly and Mathieu's[1] observation is appropriate in this connection. Intercurrent small-pox occurred in a patient after section of the sciatic nerve and the non-innervated limb remained free from eruption. These apparently conflicting facts are not so inexplicable as they would seem to be at first sight. In the third case there was total absence of all innervation, and no eruption. In the first, we may fairly suppose that there was slight vaso-motor influence, but no cerebral, and slight eruption; while in the second, the vaso-motor system was probably undamaged, but cerebral influence cut off, and the eruption was therefore violent. If this explanation be considered feasible, the eruption would be in inverse proportion to the brain influence. May not some such hypothesis as this afford an explanation for the varying severity of the eruption in the exanthemata? In confluent small-pox, for instance, the cerebral depression may be so great that the vaso-motor system is quite uncontrolled; whilst in the most malignant form this system is also paralysed, and death occurs, with no eruption except those from hæmorrhages into the cutis.

Cases of herpes zoster on the paralysed limbs of hemiplegics are on record, such as that of Duncan,[2] in whose patient zoster appeared on the thigh, and almost at the same time hemiplegia of the same side. Payne[3] gives the case of a child who had zoster of the superficial branches of the anterior crural three days after the occurrence of a hemiplegia of the same side. Charcot,[4] who quotes these cases, warns us not to draw deductions too hastily from this coincidence, by relating the case of a soldier with ulcerative endocarditis, who had a sudden paralysis of one leg and at the same time zoster of the peroneal nerve-branches of the same leg. Post-mortem, there was an embolus in the posterior cerebral artery with its corresponding area of softening, and also a clot in one of the spinal branches of the lateral sacral artery, which pressed upon one of the posterior spinal roots of the cauda equina as the vessel went through a foramen. Oppolzer, Hesselink and Chvostek,

[1] 'Archives Générales de Médecine,' 1880.
[2] 'Journal of Cutan. Medicine,' Oct. 1869, p. 69.
[3] 'British Med. Journal,' Aug. 1871.
[4] Charcot, loc. cit. p. 82.

and others, also give examples of zoster with cerebral lesions.

A review of all the above facts suggests that the relation of the cerebrum to cutaneous eruptions is mainly due to the degree of inhibiting effect exercised upon the vaso-motor centre, but there are no facts indicating from what part or parts this influence starts in the brain to the vaso-motor centre.

When we come to lesions of the spinal cord in relation to skin eruptions, not only is the material more abundant, but our knowledge is somewhat more definite, though there is still much to clear up. The difficulty here, as in the cerebrum, is to find uncomplicated cases. The nearest approach to this are three cases, in which, with bullous eruptions, the columns of Goll were especially implicated. Of these I will give abstracts. The first two were under Schwimmer's observation; the third under Dr. Meyer of Strassburg, a full abstract of which will be published in the next number of 'BRAIN.'

The first one is described as Case 13, p. 148, of Schwimmer's work, and was that of a woman, æt. 32, who five months before admission had dyspepsia, followed by pemphigus, which only lasted a short time. In her present attack the whole body, face and scalp were involved in the pemphigus, which was hæmorrhagic on the legs; diarrhœa and albuminuria set in, and led to her death. At the post-mortem by Dr. Babes there was found sclerosis of the columns of Goll; in the cervical region, the peripheral part of the postero-lateral columns were especially wasted, and there was slight cell-increase round the vessels of the posterior horns or roots, and of the postero-lateral columns. In the upper dorsal region, the right anterior horn and the external nerve-cell group was atrophied. In the upper lumbar region, of which a figure is given, the whole periphery of the cord was atrophied, the posterior roots stained strongly, and the *columns of Goll sclerosed.* The nerve elements had almost entirely disappeared, principally from a wide-spread rich-celled vascular network. In the postero-lateral columns also the neuroglia was somewhat increased.

In the second case, No. 14 of his work, a previously healthy

man had a severe chill from standing in water for some time. Soon after, the patient complained of pain and convulsive movements of the legs; he had the girdle-feeling, and then the bullous eruption broke out and soon developed into a pemphigus foliaceus. There was a temporary improvement, then a partial paraplegia came on, painful neuralgia of the upper limbs, bedsore on sacrum, and he died of exhaustion six months after admission.

Post-mortem, in this as in previous case, the cord was notably softened.

There was extreme sclerosis of the columns of Goll in the upper part of the cord up to the cervical enlargement, with neuroglia thickening. At the beginning of the dorsal region circumscribed, greyish, softened gelatinous places existed at the anterior periphery, especially the left, and the anterior horn was atrophied. At the beginning of the lumbar part there was widespread and extreme sclerosis of the posterior root-zones, with thickening and cell-increase of the vascular network, and atrophied foci of the left anterior horn corresponding with the median cell group.

Meyer's case was that of a man of 65, with previous good health. The disease began as an eczema, then it spread nearly all over the body; bullæ appeared, diarrhœa set in, and he died seven weeks from the outset of the skin eruption. Post-mortem, there was degenerative parenchymatous neuritis of many of the cutaneous nerves, sclerosis of the columns of Goll in the dorsal region, with points of sclerosis scattered through the posterior columns in the upper cervical and in the lateral columns of the dorsal and lumbar regions. The neuroglia was increased, and the walls of the blood-vessels thickened.

Scarcely less interesting, though there was no post-mortem, was Case 12 of Schwimmer, a private patient, a man aged 64, who in a street accident sustained a severe bruise of the left shoulder. After a fortnight's severe pain of the whole extremity it left a neuralgia of the upper limb, which came on at irregular intervals; three weeks later, a severe herpes zoster affected the whole extensor aspect of the limb from the shoulder to the ends of the fingers: the neuralgia gradually

diminished, and then bullæ appeared, first on the left leg and then on the trunk, coming out in crops, and attended with a rise of temperature. For the first ten days the bullæ were limited to the left side of the body, then spread to the right side, and soon became universal, including the oral mucous membranes. Abundant crops of semi-opaque bullæ, preceded by severe pain, exhausted the patient and produced great denudation of epidermis. On the sixth week of the pemphigus outbreak, paraplegia set in, with paralysis of bladder and rectum, and the patient died three months from the commencement of the herpes zoster. No post-mortem was allowed.

Interesting as these cases are, while showing that there is an association of posterior sclerosis with some cases of pemphigus, they do not even prove that the cord lesion was primary, though it was probably the case. Meyer suggests for his that the vascular obliteration produced sclerosis of the cord, and the same change in the skin-vessels predisposed the skin also to disease.

In the fourth case there can be little doubt that there was neuritis from the injury, and that the inflammation travelled up to the cord, and spread in the cord and led to the bullous eruption.

There is no disease of the cord in which eruptions are so frequent and in such great variety as in locomotor ataxy; but very often the lesions are not confined to the posterior columns, by the time they come to the post-mortem table, but there are some such in whom there have been eruptions during life. I will first enumerate the eruptions that have been noticed in ataxy:

1. Transitory erythema. { (*a*) Simple. (*b*) Nodosum.
2. Urticaria, with intense itching.
3. Papular or "Lichenoid" eruptions, probably eczematous.
4. Ordinary eczema.
5. Herpes zoster.
6. Pemphigus.
7. Pustules "like ecthyma."
8. Ulcers, superficial and deep.
9. Superficial gangrene.

10. Perforating ulcer of foot.
11. Falling off of the big toe-nail.
12. Leucoderma.
13. Petechiæ and ecchymoses.
14. Unilateral sweating.
15. Œdema.

The first nine of these lesions occur in the early stage. They are generally associated with an exacerbation of the lightning-pains, and are usually limited to the course of the nerves in whose course the pains are felt.

The Erythema, besides being transitory, is uncertain in size and position, and may be anywhere; it is especially alluded to by Charcot.[1] Erythema nodosum is mentioned by Bristowe.[2] Déjérine, Purdon, Friedrich, &c., describe urticaria. Charcot[3] had a case of a woman in whom enormous plateaux of urticaria were produced at the most painful spots during an attack of pains. In another, the right buttock was covered with herpes zoster in the course of the lightning-pains. In a third, an old woman, in whom the ataxy ran a very rapid course, two unusually severe attacks of lightning-pains were associated with very numerous pustules, "like ecthyma," which led to deep ulcerations in the course of the small sciatic and left saphena-nerve respectively; and in the first attack there was also a deep *slough*, 5 centimetres across, on the right sacral region, which took two months to heal.

The papular or lichenoid eruptions are especially common, and more or less confluent upon the legs and thighs; but may be restricted to one leg, or even to the course of a single nerve. Charcot does not appear to believe in an ordinary vesicular eczema in ataxy; but Purdon[4] found long-standing eczema of the lower extremities in the latter part of the course of ataxy; and inasmuch as several cases of eczema following neuralgia, limited to the painful area, such as that of Cavafy,[5] are known, there seems every *à priori* reason that such eczema

[1] Charcot, loc. cit. p. 76, &c.
[2] Bristowe's 'Medicine,' 3rd edition, p. 912.
[3] Charcot, loc. cit. p. 65.
[4] Purdon, 'Union Médicale,' 1878, No. 75.
[5] Cavafy, 'Brit. Med. Jour.,' July 24, 1880.

may occur sometimes and bear relation to the spinal lesion; at the same time eczema is so common, that the connection between the two must not be hastily assumed because they are present together. Pemphigus, in cases of chronic myelitis and acute meningitis, is very common. Vulpian[1] records a case with *ataxy* and lightning-pains, in whom bullæ appeared on the painful upper limbs whenever he put his hands in hot or cold water, and even spontaneously on some occasions; subsequently an elephantiasis-like condition was developed in the hands, and the finger-nails were much deformed. Gangrene is rare in locomotor ataxy; but in addition to the case of Charcot's already cited, Debove[2] records a case of a woman, æt. 44, with ataxy and lightning-pains, in whom a slough formed on the upper part of the sacrum which spread to the left buttock, and got deeper; then invaded the right buttock, and became symmetrically developed on each side of the median line, with an area of 8–9 by 6 centimetres, accompanied by *œdema* of the lower limbs. Post-mortem, there was atrophy of the posterior roots, especially at the lumbar enlargement, and sclerosis of radicular fasciculus; and in the nerves, on the border of the ulcer, parenchymatous neuritis. Perforating ulcer of the foot is now well known to be sometimes due to locomotor ataxy, although it may also be due to lesions of the peripheral ends of the nerves, the nerve-trunks, or the spinal ganglia of the affected side. Hanot and many others have demonstrated this. Falling-off of the big toe-nail occurs in ataxy, occasionally without any preceding injury, or inflammation; in one case[3] it occurred three times within eighteen months; regeneration occurs without any difficulty.

Leucoderma, as already mentioned, has been noted in connection with ataxy by Bulkley, Debove and Barthélemy.

Unilateral sweating has recently been recorded by Raynaud and Artaud.[4] It occurred on the right side of the face, head, neck and shoulder ten or fifteen minutes after a meal,

[1] Vulpian, 'Maladies de la Moëlle,' 1877, p. 57.
[2] 'Progrès Médical,' quoted by Leloir, p. 115.
[3] Joffroy and Pitres, 'Progrès Médical,' 1882, No. 8.
[4] 'Revue de Médecine,' 1884.

with no alteration of the pupil, but there was an increased flow of saliva on the affected side. Œdema has already been alluded to in Debove's case; it is especially apt to occur in the knee and ankle joint, as mentioned by Schwimmer,[1] and often disappears spontaneously.

Only one reference to the cases illustrating each cutaneous lesion has been given; but the number could, with regard to most eruptions, be easily multiplied.

The great variety of eruptions, with apparently the same kind of nervous lesion, is most striking, and we can neither predict the kind of eruption from the nature and position of the lesion, nor, with but few exceptions, can we predict the position or nature of the lesion from the kind of eruption alone. At the same time it must ever be borne in mind that, common as some kind of eruption is in locomotor ataxy, in the majority of cases with the same lesion there is no eruption at all; still the frequency is too great for the association to be purely accidental, and we can only infer that, besides the cord lesion that can be proved to exist, there are some other conditions that determine the occurrence and the nature of the eruption. Of these conditions at present next to nothing is known. The coincidence of the majority of the eruptions with lightning-pains suggested that if the position of the lesion producing them can be ascertained, that for the eruptions will also be there or thereabouts; and this, as Charcot shows, has been pretty accurately ascertained by post-mortem proof that where lightning-pains had been present, there was always sclerosis of the posterior columns; while the other lesions which might possibly account for them were absent in different instances, viz. atrophy of the posterior roots, posterior spinal meningitis, sclerosis of the posterior cornua, and irritative lesions of the spinal ganglia or peripheral nerves. A most striking instance is a case of Charcot's,[2] in which a woman had suffered severely from lightning-pains for fifteen years, but with no motor symptoms; at the autopsy the only appreciable lesions were in the posterior columns, where there was commencing sclerosis, with no alteration of the nerve-tubes, imbedded in the thickened neuroglia.

[1] Loc. cit. p. 181. [2] Loc. cit. p. 78.

The posterior root zones, Charcot thinks, consist of sensory fibres, a certain number for the performance of reflex acts, and others which have a more or less direct influence on the nutrition of the skin. These last, he suggests, are less easily irritated than the sensory fibres, and this may account for the frequent absence of eruptions, on the one hand, and for their varying intensity where present, and so for the existence of erythematous, papular, vesicular, bullous, pustular or gangrenous eruptions. It must, however, be borne in mind that the last three eruptions are also frequently the result of the loss of nerve influence, rendering the tissues unable to resist noxious external influences. While therefore a rather strong case is made out in favour of the postero-external column being largely concerned in ataxic, and many other skin eruptions due to cord disease, we cannot say that they are the sole arbiters in this matter. The cases first related point to the columns of Goll having some influence; of the posterior grey cornua we can say nothing; of the antero-lateral columns and anterior grey cornua, the evidence is against them for the most part. The few cases in which there have been eruptions in association with diseases in which the anterior part of the cord is mainly concerned, have been always open to the suspicion of their being more or less complicated. Thus Balmer [1] gives three cases of progressive muscular atrophy with slight loss of sensibility, in the course of which pemphigus supervened.

If Gowers's [2] supposition, that there are some sensory fibres in the tract in front of the crossed pyramidal tract, as supported to some extent by animal experiments of Woroschiloff and Ott, and by Gowers's own case and that of Hadden [2] (in the last, unfortunately, the clinical history is unknown), this column may have to be considered with regard to a possible influence on the skin; nevertheless it remains that the posterior columns of the cord have a very important influence upon the nutrition of the skin; and the more or less direct production of cutaneous eruptions. Before concluding the consideration of the spinal-cord influence on the skin, mention must be made of the numerous eruptions recorded in associa-

[1] 'Archiv für Heilkunde,' 1875, p. 317.

[2] 'Diagnosis of Diseases of the Spinal Cord,' pp. 13 and 14, 1st ed.

tion with spinal meningitis, especially vesicular eruptions, such as the herpetic and pemphigoid. Erb[1] also enumerates, roseola, petechiæ, erythema, urticaria and erysipelas. With the exception of erysipelas, which was probably either a purely accidental concurrence, or an error of diagnosis on the part of the recorder, all the lesions are similar to those found in locomotor ataxy, and they are doubtless explicable by the irritation set up in the posterior columns, on the one hand, or in the issuing nerves, on the other. Erb also states that herpes and bullæ are frequent in slow compression of the cord.[2] As in the brain, vaso-motor disturbances may be set up in connection with cord disease without any visible lesion. Thus in acute ascending paralysis, in which lesions have hitherto been searched for in vain,[3] Eisenlohr had a case with transitory œdema, redness over the joints and profuse sweating.

The lesion for acute decubitus cannot be localised more definitely than by saying it is found especially in acute central myelitis; and that it may occur in ataxy has already been shown. Its occurrence in cerebral lesions, hæmorrhagic, softening and traumatic, is well known, and it is then always on the paralysed side, and pressure, though often very slight, seems to determine its position.[4] Brown-Séquard has shown that in two-thirds of the cases the cerebral lesion is on the right side. I am not aware of any case being recorded showing that the cord was free from secondary change in such cases.

Coming now to the cerebral and spinal-nerve ganglia, the ground is somewhat firmer. It was here that Bärensprung first demonstrated anatomically, what had long been urged on clinical grounds by Rayer, Charcot, Parrot and Danielsen,[5] the neurotic origin of herpes zoster, and while it was the first to be thus proved, it is also the type of neurotic dermatonoses. Bärensprung demonstrated that in his case there was inter-

[1] 'Pathological Society's Transactions,' vol. xxxiii., p. 21.

[2] Ziemssen, vol. xiii. art. "Spinal Meningitis."

[3] Quoted by Erb in Ziemssen, loc. cit.

[4] Quoted by Bastian, 'On Paralysis from Brain Disease,' p. 210.

[5] Danielsen, it must in justice be said, had proved a neuritis of two intercostal nerves in a case of zoster, before Bärensprung showed that the ganglia were at fault.

stitial neuritis of the spinal ganglion, and of the trunk of the nerve coming from it. This observation has been abundantly confirmed for the majority of cases by many subsequent observers, and the presence of the same kind of lesion, namely, inflammation of the Gasserian ganglion,[1] has been proved in cases of herpes frontalis; but Bärensprung's assumption that all cases of zoster were dependent on lesion of the nerve-ganglion is now known to be true for the majority of cases only, and not for all.

As already mentioned, herpes is common in cord disease, especially central myelitis, ataxy, and meningitis, and in a few instances the ganglion has been examined and found to be quite normal. Werdner[2] in one case found a lesion of the posterior spinal roots, while the cord and spinal ganglion were healthy. Strangely enough, the posterior roots are healthy as a rule when the ganglia are affected, which is contrary to what would *à priori* be anticipated.

The cases of zoster with cerebral lesions have been alluded to, and they are too open to fallacy to argue from. There are also cases which give clinical evidence that the ganglia are not always at fault; thus cases of double zoster are very unlikely to be due to inflammation of both ganglia, and as many are of syphilitic origin, probably a local meningitis would account better for them.

There would even seem to be a possibility of some cases being due to reflex irritation. Jewel[3] relates a case of zoster cruralis as a consequence of severe uterine disease, the zoster and neuralgia disappearing when the uterine trouble was removed. Lendel[4] relates cases of zoster from poisoning by carbonic oxide gas, which he ascribes to a neuritis toxica, while Schwimmer regards them as reflex. Injuries and neuritis of the trunk of the nerve have frequently given rise to zoster, and while it is very likely that the zoster was directly due to this neuritis, the ganglia have never yet been proved to be normal in such cases. The periphery of the nerves at the

[1] By O. Wyss and Horner, quoted by Leloir, p. 152.
[2] Quoted by Leloir, p. 154.
[3] 'Trans. Amer. Neurological Assoc.,' Nov. 1875.
[4] Lendel, 'Archives Génér. de Médecine,' 1865.

skin has not been examined, but the presence of defects of sensibility in some cases is suggestive that the cutaneous nerves may be affected, at all events.

There is no doubt that peripheral irritation will produce herpes. I have seen a case in which inflammation at the root of a tooth, leading to abscess and a sinus, excited severe attacks of herpes round the sinus, which ceased when the tooth was removed. Mr. Pearce Gould had a case of caries of a rib, with recurrent herpes. Lesser[1] relates a case of a young man who had the nerve destroyed of the last molar but one in the lower jaw. Two days after, groups of vesicles, accompanied with pain, appeared on the chin and lower lip in the course of the left mental nerve; five days later the first crop had dried up, but three fresh groups of vesicles came out on the left cheek and one vesicle on the left auricle. Sensibility was normal. Lesser explains this as follows: inflammation spread from the branch of the alveolar nerve to that nerve itself; thence to the mental nerve, which is a direct continuation of the alveolar nerve; then advanced centripetally, and then down again, affecting the auricular temporal nerve. If this explanation be true, and it seems the most probable, the eruption in the tract of the mental nerve was produced by an inflammation of the nerve without the implication of the ganglion; and in my case, and that of Mr. Gould's, this seems also have been the case: anatomical proof of this cause of herpes without implication of the ganglion is still wanting. The lesion of the ganglion is not always the same. Kaposi[2] had a case of herpes frontalis, in which he found hæmorrhage into the Gasserian ganglion. Wyss and Satter have had similar cases. Hæmorrhage into the cauda equina with crural herpes has also occurred. Charcot's case of herpes from an embolus in a branch of a sacral artery has already been mentioned. He also relates a case of carcinoma of the vertebræ with herpes; while Ollivier[3] found cancer of the pleura and lung on the same side as the herpes.

Gunshot and other injuries, as related by Weir Mitchell,

[1] Virchow's 'Archiv,' vol. xciii. 1883.
[2] Kaposi, 'Hautkrankheiten,' p. 322, 1883.
[3] 'Wiener Med. Wochenschrift,' 1877, No. 25-27.

Morehouse and Keen, &c., are other causes of neuritis only, as the eruption was always some time after the injury; herpes may thus occur as a result of any kind of irritant in the ganglion or any part of the issuing nerve. It has been anatomically proved that the lesion may be either in the cord, the posterior root, or the ganglion, and it is probable that a lesion of the cutaneous nerves alone is capable of producing it. Reflex herpes is of more doubtful existence. It has never been shown that it may occur from uncomplicated cerebral lesions, those cases associated with cerebral lesions being always more or less open to the possibility of other nerve defects.

Similar instances of the same eruption being due to lesion of different parts of the nervous chain are seen in pemphigus leucoderma, and other pigmentary alterations, in perforating ulcer, &c.

Cutaneous disease from lesions of the branch or branches of nerves below the ganglion have been especially described by Paget, Weir Mitchell, Morehouse and Keen, and their observations have been confirmed with very little addition by many others. Leprosy also, by its implication of the nerves, affords us information in the same direction; but even here, as in more central disease, the total absence of defects in the nutrition of the skin where the same kind of lesion in the nerves existed is quite as striking as the occurrence of the eruptions. The following observations are mainly from Mitchell's well-known work. The nutritive and other defects that have been recorded are:—

1. Erythema, persistent and abscess-like.
2. Herpes.
3. Bullæ.
4. Ulcerations. { Simple. / Perforating.
5. Eczema.
6. Glossy skin.
7. Defects of nails.
8. „ hair.
9. „ pigment.
10. Chronic œdema, like Elephantiasis.
11. Ichthyosis-like condition.

In complete division of a nerve, there is with the general atrophy "œdema, with finally discoloration of the dry and thickened skin, the ragged epithelia hanging in patches."[1] Some ulcerations also occur, but these are the result of injury, often slight, which the non-innervated tissues are unable to resist. Bullæ are rare in complete section of a nerve. Mitchell never saw them in the small number of cases where there was section of all the nerves supplying a part; yet, as Arnozan points out, the bullæ come where the anæsthesia is complete. Nevertheless, bullæ do come out where the function of the nerve is quite destroyed; as in cases of anæsthetic leprosy, where both paralysis and anæsthesia are complete, bullæ come out from time to time on exposure to cold. Like the ulcers, therefore, they require a local irritation to produce them. These bullæ often leave slow-healing ulcers; yet, as a rule, after total section of a nerve, wounds heal fairly well, if put in proper condition. Where the nerves have been injured in a slight degree, the eruptions are much more numerous and important. The erythema that is sequential to nerve injury lasts long and simulates an abscess. This form of erythema is regarded by Paget[2] as characteristic of nerve injury, a statement which is confirmed by Charcot and Brown-Séquard. Probably this is the same condition that is described by other authors as erythema nodosum, which occurs in cases of neuritis. Herpetic eruptions are frequent after injury to nerves. Brown-Séquard,[3] mentions a contusion of the internal cutaneous nerve followed by herpes in the affected region. In a healing wound over an injured median nerve, Mitchell witnessed ulcerated matrices of nails and vesicular eruption. There is also Rayer's[4] well-known case, reported by Charcot, of gunshot-wound of the thigh, followed by neuralgia and the outbreak of repeated crops of herpes at the painful spots. These cases could easily be multiplied.

Bullæ are on much the same footing as herpes. In one of Charcot's[5] cases, abscesses and the necessary incisions injured

[1] Weir Mitchell, loc. cit., p. 152.
[2] Paget, 'Med. Times and Gaz.' 1874.
[3] Weir Mitchell, loc. cit. 153, quotes this.
[4] Ibid.
[5] Ibid.

the nerves of the arm, and was followed by neuralgia and other symptoms; with bullæ, which formed in a few hours, grew as large as a nut, and left a quickly healing ulcer. These came chiefly about the joints of the first three fingers. Mitchell quotes several other cases in the same place of this kind of lesion.

In non-tuberculated leprosy, among the early symptoms are shooting-pains, with the eruption of bullæ upon the fingers and toes. These differ from the bullæ of the advanced anæsthetic leprosy in being spontaneous, small, numerous, and occurring on a painful surface; while the others are large, solitary, on an anæsthetic part, and usually excited by local causes.

Ulceration generally follows the bullæ in partial injury to a nerve, and is not due to local injuries like ulceration after total section; sometimes, as Hutchinson states, it begins with a whitlow-like condition, with considerable inflammation. The whitlow is painful or not, according to the locality in an anæsthetic or hyperæsthetic area. Mitchell[1] also describes eczematous conditions either on healthy or atrophied skin. Cavafy's already cited case confirms this; and Arnozan relates a case of Brouardel's, where a man of 60 had a contusion of the shoulder, followed by pains shooting down the arm, and in forty hours an eczema, limited to the radial nerve, becoming rapidly confluent and getting well in a fortnight.

The condition known as "glossy skin" appears to be always the result of neuritis, traumatic, gouty, or rheumatic, &c. It begins some little time after an injury, and is often excited by an inflammation supervening on the original wound. Thus Mitchell relates the case of an attack of erysipelas supervening upon a wound of the radial, followed by the peculiar "causalgia," glossy skin and eruptions; the patient having previously had partial loss of motion and sensation. It is in glossy skin that eczema is so frequent and often very persistent.

Mitchell gives a case where the glossy skin was associated with an antero-lateral sclerosis; intense neuralgia, motor palsy of legs, with contracted toes and ankles; no loss of sensation; neuralgia of arms and hands; contraction of fingers; no palsy

[1] Loc. cit., p. 154.

of upper limbs; hyperæsthesia of palms; causalgia and glazed redness of ulnar side of palms. It is evident that there were other secondary changes besides the antero-sclerosis, but whether it extended down the nerves of the limbs cannot be determined. There were nutritive changes in the nails and teeth in this case.

"After total section of a nerve the nails are apt to become clubbed, and in rare cases to suffer from painless whitlows" (Mitchell):[1] growth is also retarded, but not stopped, and nails may even be reproduced. Furrowing and white spots are common.

In partial destruction of a nerve, painful ulceration of the matrix may occur; and when there is glossy skin, the nails curve both longitudinally and transversely with thickening of the cutis beneath; or else the line of union of the nail and skin begins lower down under the nail, and the border is ragged and notched. In the toes the nails are less deformed, but a painful and recurring ulceration occurs at their angles.

In cases without glossy skin, the nails may be only dry, scaly, very thin, cracked and fragile; or furrowed, pitted and discoloured. In hemiplegias nail-growth is arrested.

The hair falls off, or is very scanty and poor in glossy skin; in neuralgias and injuries of the trigeminal it often becomes white or falls out temporarily; in some cases the blanching persists. In a traumatic neuralgia, Ponteau saw the hair grow large and hard, with a tendency to stand erect.

Larrey describes similar cases on hyperæsthetic skin.

Crampton witnessed a case in which the arm became thickly hairy in the bend of the elbow after a lancet wound[2] which injured the musculo-cutaneus nerve in "bleeding," and is supposed to have set up a neuritis. Section of the nerve gave partial relief, but the skin recovered after salivation for an attack of pneumonia.

Schwimmer[2] gives a case of a wound of the temple, followed by an alopecia patch in the temple and upper lip of the same side.

In general, there is loss of hair after division of a sensory nerve, and increase after division of the sympathetic.

[1] Hutchinson, quoted by Mitchell, p. 153.

[2] Schwimmer, loc. cit. p. 222. He also quotes the other cases from Mitchell.

With regard to pigmentation defects, Schwimmer[1] gives a case of bilateral herpes in which in the course of the affected nerves white patches appeared; while he quotes O. Wyss's case of loss of pigment from disease of the second division of the trigeminal.

In another of Schwimmer's cases after the herpes, spots and striæ appeared on the back of the forearm in lines to the back of the hand and fingers. The ganglia may have been implicated in these cases.

A chronic œdema producing an elephantiasis-like condition is sometimes induced.

Mitchell gives the case of a bullet-wound of the arm near the axilla; there were darting-pains down the limb; the skin was dry, scaly, and yellow, and an elephantiasis-like condition of the thumb and first two fingers and the radial side of the back of the hand; there was slight œdema of the ulnar side of the palm and back of the hand.

An ichthyotic condition is occasionally developed after an injury to a nerve. Leloir gives a case that was under M. Trélat.—A man fell and received a contused wound, dividing the ulnar nerve incompletely; the ulnar region became anæsthetic. This lasted a short time, but during that period the affected region was in an ichthyotic condition; the skin was thickened and peeled, and looked like "serpentine ichthyosis." As sensation returned, the skin improved, and three weeks after the injury, sensation and the skin were normal.

In another case this condition supervened after a wound of the nerve of the index-finger. There was recovery in a short time of sensation and health to the skin. In paraplegics this condition also occurs. Leloir gives cases, and figures one with ichthyosis of the legs in a painter, who suffered from lead-poisoning and cerebro-spinal disease. The cerebral arteries were thickened and atheromatous. The spinal cord was not examined.

Congenital cases of ichthyosis hystrix in the course of nerves are well known, but they appear to be in a somewhat different category to ordinary ichthyosis.

These cases of acquired ichthyosis probably differ from the

[1] Schwimmer, loc. cit. p. 211. Leloir, loc. cit. p. 59.

ordinary congenital form, but there is no other term that would so well convey the appearance.

That perforating ulcer results from atrophic degenerative associated with interstitial neuritis, has been established by many observers. Poncet,[1] Duplay[2] and Moralt, Michaud, Sonnemburg[3] and Savory,[4] and Butlin have all made valuable contributions in this direction. In anæsthetic leprosy it is common, and Déjérine and Leloir have made researches upon this subject.

The ganglia and the peripheral nerves have also been found in a condition of atrophic parenchymatous neuritis in perforating ulcer.

The original part of Leloir's Thesis is mainly concerned with the lesions of the cutaneous nerves. To go fully into the subject would be, but to reproduce his very careful and valuable work.

That there should be lesions of the cutaneous nerves in eruptions of the skin at the site of the eruption might be anticipated as a probable event, the nerves being involved secondarily to the skin lesion; but research shows that the nerves are not so easily affected, and when therefore they are found to be diseased, it is the more significant. Déjérine and Leloir have examined four cases of psoriasis, three of tubercular lupus, one of lupus erythematosus, five of ecthyma, (three of them occurring in typhoid fever, and two in cachectic individuals), a case of ulceration of the big toe, with melanodermia and epithelioma of the lip, and all with negative results. Balzer has never found lesions of nerves in syphilides, psoriasis, lupus, or epitheliomas; Chambard has not in gummata; Poncel in the neighbourhood of abscesses; and Quénu where the pneumogastric traversed cheesy glands; nor Vulpian in gangrene. Weir Mitchell, Cornil, and Ranvier, &c., confirm these observations.

Leloir therefore considers that he is entitled to assume, that when cutaneous lesions are found they are primary.

[1] 'Mémoires de Médecine Militaire,' 1864, tome xii.
[2] 'Archives de Médecine,' 1872.
[3] Sonnemburg, 'Deutsche Zeitschrift für Chirurgie,' 1876.
[4] 'Medico-Chirurgical Trans.,' 1879, p. 360, &c.

In all, the character of the lesions is the same, viz. atrophic parenchymatous neuritis.

The nerves were found affected thus in three cases of vitiligo; two by Leloir, one by Déjérine.

In one of these there was syphilis, but no central lesion proved or probable on clinical grounds.

The second and third cases were fatal cases of phthisis. Here also no central lesion was to be anticipated, but in none of them was there any examination of any other parts of the nervous system.

Lesions were found in two cases of ecthyma. One was a general paralytic; the other with cerebral hæmorrhage on the right side, and the ecthyma on the left side.

In one serpentine ichthyosis case, pemphigus was also present, and the centres were not examined.

In three cases of pemphigus—

1. Examined by Déjérine, has already been alluded to. The patient was a general paralytic, and peri-encephalitis and lateral sclerosis of cord were found

2. Examined by Quinquaud. There was spinal hemiplegia sclerosis of the lateral column encroaching upon the anterior and posterior columns.

3. By Leloir. A woman aged 72, very cachectic on admission to the hospital, died from exhaustion from "bilious vomiting." The centres were not examined.

4. Jarisch[1] also has found the cutaneous nerves diseased in a similar way in a woman who had so-called herpes iris. He also found changes in the central, lateral, and posterior parts of the cord.

Thus, out of the ten cases, five had certainly central disease. In the other five the centres were not examined. The complication of the ichthyosis with pemphigus reduces its value somewhat, and we cannot argue from it. In the case of the old woman who died with "bilious vomiting," the absence of central disease can scarcely be affirmed positively. This leaves the cases of vitiligo. In these there was probably no actual central lesion; though since we know that even

[1] 'Vierteljahr. für Dermat. und Syph.' 1880.

mental disturbance will produce vitiligo, it would be rash to say that some central influence had nothing to do with it.

In the other five cases it would seem probable, to say the least, that the central lesion had as much to do with the peripheral nerve lesion as that lesion could have with the eruption; and it is arguable that, though lesions of the skin do not affect the nerves of the part as a rule, they may do so when the protecting influence of the nervous centres is in abeyance, even if not acting adversely. But since we see that so many skin lesions are conduced to, if not actually produced by, a defective link in the nervous chain of brain cord, ganglia or trunk of a nerve, it seems probable that a defect at the peripheral end, viz. at the cutaneous nerves, might produce skin disease; but nevertheless, in the face of the above considerations, we must pause before we jump to the conclusion, that these lesions of the cutaneous nerves occurred independently and produced lesions of the skin.

There is, however, one case examined by Leloir and Déjérine which is less open to fallacy, and in favour of Leloir's contention.

In this, a woman, the subject of chronic rheumatism and muscular atrophy, with albumen in the urine, with dulled intellect and cachectic appearance, æt. 49, died twenty-three days after admission—five days before death three sloughs formed on the lower part of the sacrum, of the size of a crown. The cutaneous nerves were in a condition of parenchymatous neuritis in the neighbourhood of the slough, the intramuscular nerves were similarly affected, the spinal roots and the spinal cord throughout its whole length were quite healthy. The brain and meninges were quite healthy to the naked eye. Still the cerebral influence could scarcely have been healthily exercised. In two cases, one of a woman with hemiplegia, in which cutaneous gangrene followed the application of a mustard poultice; the other, of a cachectic woman attacked with cutaneous and visceral gangrene, Leloir found the cutaneous nerves were quite healthy. These give more force to the first case. In Leloir's Case XXX. of patches of cutaneous gangrene in a hysterical girl, had the case not been seen by such able observers as Hardy, Vulpian, and Lailler, I

should certainly from the description have come to the conclusion that the lesions were self-induced and nervous only in reference to her mental condition.

His Case XXIX. has been already alluded to under gangrene with ataxy, and though the cutaneous nerves were affected, few will allow that they were primarily the cause of the sloughing, as Leloir contends.

Quinquaud, quoted by Percheron,[1] has published a case of general exfoliative dermatitis, with parenchymatous neuritis of the cutaneous nerves, supplied by the intercostal and sciatic with diffuse myelitis; and Lancereaux[2] has also published a similar case where the nerve centres are involved.

These facts certainly suggest the probability that the cutaneous nerves do not give way until the central influence is weakened.

Finally, the sympathetic and its lesions remain to be considered. Although the number of cases affording clinical evidence of the influence of the sympathetic upon the skin is considerable, there are but few cases where an actual lesion has been demonstrated, and even in some of these the proof is indirect.

The effects are shown in,

1. Congestion.
2. Pallor.
3. Pigmentation.
4. Sweating.
5. Increased growth of hair.

The close relationship of the sympathetic with the cervical enlargement of the cord is universally acknowledged, so that lesions here with vaso-motor phenomena may fairly be accepted as demonstrated lesions of the sympathetic. These two cases of Rendu[3] illustrate opposite effects.

The first was a case of dislocation of the 6th cervical. There was paraplegia; the pupils were contracted; the face was hot and turgid, although the skin of the face and neck were of a

[1] Percheron, 'Thèse de Paris; dermatitis exfoliatrice.'
[2] Lancereaux, 'Traité d'Anatomie Pathologique,' tome i. p. 275.
[3] 'Archives générales de Médecine,' 1869, II.

violet tint. In the cheeks and nose the smallest capillaries were visible, the ears were of a deep red; the rest of the body was normal.

In the second case there was a fracture of the two last cervical and the first dorsal vertebræ, with generally paralysis and extreme pallor of the whole surface of the skin.

Mitchell[1] gives a more direct case. The right sympathetic was wounded by a bullet. Among other symptoms the man had contracted pupil, with unilateral flushing of the right side of the face on exertion.

The erythematous and urticarial lesions that have so often been spoken of in relation to various nervous lesions are doubtless immediately due to the action or inaction of the sympathetic, but we have no more positive evidence of sympathetic defect in these cases. In a case of universal acute eczema, Marcacci[2] found changes in the sympathetic.

Such doubtful lesions as hyperæmia of cervical ganglia in unilateral sweating (Ebstein), pigmentation of ganglia in pemphigus, universal atrophy of the skin and sclerema (Schwimmer) do not count for much. Indeed, the same pigmentation was found in a healthy man killed by accident.

With regard to pigmentation, out of 33 cases of Addison's disease,[3] in 21 there were lesions of the abdominal sympathetic. The changes were partly from thickening and partly from atrophy, and often only affected single ganglia of the cœliac plexus. There has also been found fatty degeneration, caseous degeneration, small cell infiltration, in single ganglia, &c. The proof of the dependence of the pigmentation in Addison's disease upon the sympathetic is therefore pretty strong; the absence of pigmentation in some other cases where the ganglia were diseased being only in accordance with the other lesions of the nervous system and the skin. The influence of the sympathetic upon sweating is generally acknowledged.

In Traube's[4] case there was a tumour in the cord half an inch

[1] Loc. cit. p. 318.

[2] 'Giornale Italiano delle Malatie della pelle,' 1878, June number.

[3] Guttmann in Eulenburg's 'Real-Encyclopädie,' I. Bd. s. 128.

[4] 'Gesammelte Beiträge zur Path. u. Phys.,' Berlin 1871, ii. p. 1012.

below the medulla oblongata, and profuse sweating a few days before death.

Claude Bernard's experiments showed, that paralysis of the sympathetic ganglia produced hyperidrosis. The dependence of the sebaceous secretion upon the sympathetic is only inferrible from analogy; but Oscar Simon divided the nerve supplying the oil-gland of the duck, stimulated the gland end, and saw the oily droplets poured out.

The relation of the sympathetic to the sensory fibres is shown by various experiments and facts in regard to the induction of sweating.

Brown-Séquard produced sweating by the direct excitation of sensory nerves.

Kendall and Luchsinger[1] produced sweating in an amputated limb, twenty minutes after the operation, by irritating the nerves of the stump.

Dieffenbach found in an artificial nose that sweat returned when sensation was recovered. Whether the centre for the regulation of the sweat is in the medulla oblongata or cord is doubtful, but there is no doubt that the path of conduction is in the sympathetic.

The hair-growth is certainly influenced by the sympathetic; in rabbits, division of the cervical sympathetic is followed by increased growth of hair. The cases of injury to nerves affecting the hair already gone into, doubtless act through the sympathetic, but no lesion is provable.

The actual anatomical facts, therefore, with regard to the sympathetic are very meagre; but it is not by any means to be inferred that the sympathetic influence is not of high importance with regard to skin lesions, and indeed it is not improbable that the rest of the nervous system acts upon the skin almost entirely through the sympathetic.

The general statements that may be made from a consideration of the preceding facts are as follows:—

That less serious consequences ensue from cutting off the nervous supply than from irritant or inflammatory lesions of the parts of the nervous system that affect the skin.

[1] 'Archiv für die gesammte Physiologie,' 1876, p. 212.

That the kind of eruption produced by the nervous system varies greatly, often without any evident reason, when the nervous defect is apparently the same in place and kind.

That the same eruption may owe its origin to any defective link in the nervous chain from the centre to the periphery.

That the same kind of nervous lesion that at one time appears to excite an eruption or other nutritive defect in the skin, even more frequently produces no change in the skin whatever.

The lesions other than atrophy that result when innervation is abolished are often traceable to external injurious influences which the tissues, when unprotected by the nervous system, are unable to resist; but we know nothing of the conditions that determine the nature of the eruption or other skin defect when the nerve lesion is irritative, nor what it is that determines whether there shall be any eruption, or none at all. This uncertainty of effect suggests that the nervous influence is an indirect one.

The cerebral effect appears to vary according to whether its control over the vaso-motor centre is increased or decreased and to the secondary changes it induces in the cord. No localising lesions have yet been found for its influence on the vaso-motor centre.

In the spinal cord the fibres that preside over the nutrition of the skin are bound up with the sensory fibres, and reside therefore mainly in the posterior columns.

That outside the cord the path is by the posterior roots, the spinal ganglia, and the sensory fibres, and that lesions of any one or more of these may lead to changes in the skin.

In drawing up this paper my aim has been to place before the readers of 'BRAIN,' in a small compass, the facts that pathology affords with regard to the relation of the nervous system to the skin, and to give a *résumé* of the most definite cases which are collected in the works of the authors at the head of my paper.

I have omitted a large number of cases which would only

have been a repetition of the same fact, and also many others which, though interesting, were too complicated to be of service in determining what part of the nervous system was responsible for the skin lesion. Although I have not brought much new material together, I trust that a restatement of the facts with somewhat different grouping may be useful to those who are interested in the subject. I leave for others the discussion of the different theories that have been advanced to explain the relationship of the nervous system to the skin.

THE NERVOUS DISCHARGE.[1]

BY CHARLES MERCIER, M.B.

We eat and we work. These are the two great functions of the human organism—of all animate beings. All other functions, save the reproductive, are subservient to these two, and these two reciprocally subserve each other. We cannot work without eating; we work in order to eat; and we cannot eat unless we work.[2]

What happens when we eat? Food is taken into the body, and therein undergoes various transformations. It is dissolved, recombined, absorbed, circulated, combined into tissue, oxidised, decomposed, dissolved and ejected. We can sum up all these changes in one word—rearrangement. The atoms and molecules of the substances forming the food are rearranged into solutions, rearranged into blood-plasma, rearranged into tissue, and finally rearranged into combinations which are of

[1] Not long ago an experienced alienist expressed to me in conversation his thorough disbelief in the existence of a nervous discharge. My surprise diminished when I found that the discharge of which he spoke was of the nature of a discharge of pus. Such a conception of the nervous discharge is so discrepant from that which is usual among neurologists, that I could not but look upon its existence as a reproach to Neurology. On search, I could find no statements of the nature of the nervous discharge, except the original exposition in Mr. Herbert Spencer's 'Principles of Biology and Psychology.' As these works are not, perhaps, universally in the hands of alienists, nor even of neurologists, I have thought it might be useful to set forth the nature of this discharge as now conceived by those who are most engaged upon the subject, in language as familiar and as free from technicality as the nature of the topic will allow. While following Mr. Spencer in the main, I have not scrupled to pursue the matter into further detail, and to make considerable additions and modifications which he might not approve. To me, therefore, belongs the responsibility for the views herein contained, while to Mr. Spencer is due the credit.

[2] The term "work" is used, not in its limited sense as equivalent to labour, but in the larger sense of producing movement. When I lift my hand to my mouth, there is work done which can be measured in foot-pounds.

no further service to the organism, and which are then expelled. The whole history of the food and the oxygen that are taken into the body consists in successive rearrangements of composition and changes, that is rearrangements, of place, from the time they enter the body until the moment they leave it. All the functions of mastication, deglutition, digestion, absorption, circulation, assimilation, nutrition, growth, secretion and excretion, are parts of a single process—the rearrangement of matter within the organism.

How do we work? By moving our muscles. This is the one and only means by which we can alter the world outside of us. When a muscle contracts, a considerable movement of some part of the body occurs. But even when the muscle has repeatedly contracted, there does not result a large rearrangement of matter within the organism. A little of the muscular structure has been used up; a little new material has been introduced; but at the end of the exertion the arrangement of the muscular structure is but little different from the arrangement that it had at the beginning. Where then has all the motion come from, seeing that motion no more than matter can originate out of nothing? Obviously, the molar motion of the muscles and limbs has been supplied by the transformation of molecular motion. There has not been a large rearrangement of matter, but there has been a large redistribution of force within the organism. The redistribution of force in great quantities and to great distances, without any discernible rearrangement of matter, occurs in every electric-light lead and in every telegraph-wire. The contraction of the muscles is set up by the delivery of a shock of force at a nerve-end—another redistribution of force. This shock has travelled—has been redistributed—from a nerve centre. The nerve centre was provoked to emit its force by the arrival of a shock from some other region of the body, and we may continue to trace the communication of force to one part of the body from another, until we ultimately find that the whole process was set going by a force impressed on the organism by some moving thing in the outer world. At every step except the first, and in some cases the last, we find that the passage, or communication, or redistribution of force is the

conspicuous, important and permanent part of the process; while the rearrangement of matter is trifling, or imperceptible, or temporary.

All the bodily organs and all the bodily functions may be divided into two great groups, according as they subserve the rearrangement of matter or the redistribution of force. The latter is, from the physical point of view, the prime function of the nervous system, of which the muscles, bones, &c., may be regarded as mere appendages.

Redistribution of force means, as we have just seen, the communication of force from one place to another. Matter is communicated from one part of the body to another in the blood-vessels, in which it is continually circulating. Force is communicated from one part of the body to another along the nerves, in which it also is continually circulating. Little waves of force pass along the nerve-fibres, at a rate of about 90 feet per second. The matter of the nerve does not appreciably change its place, the force alone travels, just as a wave passes through a fluid in the way familiar to every student of physics. But redistribution of force means something more than communication of force from place to place, and something more than this is included in the physical functions of the nervous system. Were communication of force the sole function carried on by the nervous system, we could never have a muscular contraction except upon the application of a proportionate stimulus to the surface of the body. The muscles are not always in full action. They contract when a shock is delivered into them by a nerve, and only then. It is obvious that we could not always depend upon having an amount of force, of precisely the requisite amount to cause a muscle to contract, applied to the body at precisely the moment at which it would be convenient for us to have such a contraction. Yet without such a stimulus applied just in the nick of time, no contraction could take place if the nervous system were solely an apparatus for the communication of force. In order that muscles should be made to contract at the right time, there must be a store of force accumulated somewhere in the body and capable of escaping from time to time in such quantity as to set up

contractions in the muscles, without immediate and direct provocation from outside the body. To form such a store is the function of the grey matter. The function of the nerves is to form channels of communication along which force may be transmitted from one part of the body to another. The function of the grey matter is to act as a receptacle or reservoir for the storage of force, and to liberate this force at appropriate intervals, in appropriate quantities and in appropriate directions.

The conception of the storage of force is familiar to every one in the leading case of coal. Every one knows that this homely subtance derives all its value from the immense store of force it contains, and the ease with which this force can be liberated when we want it; and it is not difficult to form a moderately clear concept of the means by which the force is held in thrall. The classical illustration is the raising of a stone. When a stone is raised, a certain amount of force is used in raising it. If it is dropped, it strikes the ground with a force equal to that employed in lifting it. If it is again lifted and placed on a shelf, the force employed in lifting it becomes latent or potential. It is not manifested in any way, but it is still in existence, for however long the stone may remain on the shelf, if it is at last pushed over the edge it falls to the ground, and expends in doing so an amount of force equal to that employed in raising it years, or it may be centuries before. When the stone was placed on the shelf, the force employed in lifting it was rendered latent or placed in store; when the stone fell, the force was liberated. Now instead of the earth and the stone—one vast mass and one small one—imagine two very small particles, one of carbon and one of oxygen, and for gravitation substitute chemical attraction. So long as the carbon atom and the oxygen atom are kept apart, there is force potential or latent or stored up. When they clash together this force is liberated.

The method in which force is stored up in the grey matter is not, we suppose, quite like the case of the stone on the shelf, nor that of the restrained chemical action. Instead of a stone lifted, imagine a brick set up on end. To do this requires the expenditure of force. Now if the ground is shaken, the brick

falls, and liberates in falling a force equal to that expended in raising it. Again, imagine a brick set on end with another brick placed across the top of it. The upper brick can now be knocked off the lower and the force which raised it be liberated, while the lower brick is left standing, with the force that raised it still in store. It is evident that a brick balanced on the top of another one will be displaced by a gentler shake than is required to knock down the single brick. A third brick may be placed on the top of the second, and this may be displaced by a still gentler shake, and its fall will liberate a still smaller part of the force employed in putting the three together. Now suppose more and more bricks are added, until we have quite a complicated structure composed of loose bricks. It is easy to see how readily a top brick could be knocked off—how slight a force would be necessary to upset it, how readily the fall of one would conduce to the fall of more, and how little the fall of a few of the topmost bricks would modify the entire structure, especially if we suppose that they do not fall off the pile, but merely from their ends on to their sides. Now if we imagine these bricks to be connected to the pile by elastic bases, so that when they have been knocked down they will slowly rise again, with perhaps a little help, to their erect position, we shall have a diagram which will represent very roughly what we suppose to be the mechanism of the nervous discharge.

We know that the grey matter of the nervous system has an extremely complex molecular condition. Each single molecule has been calculated to contain nearly one thousand atoms. These atoms are united together in twos and threes, in dozens and scores; several of these clusters are united together to form a larger group, which is again combined with others in several links of cross-relationship. The whole molecule is therefore a very complicated structure, and may contain many atoms and groups of atoms that stand in the position of top-bricks.

A structure so complicated is easily disarranged. When a disturbing force impinges against it, the weakest ties between the atoms and groups of atoms will give way, and some of the atoms will fall into new positions, just as the top bricks fall into

new positions when the pile of bricks is shaken. Now mark, that in building up the atoms into the complex arrangement found in the molecule, force has been employed—has become latent—has been stored. And when the atoms are shaken into simpler positions, just so much force is liberated as would suffice to build them from this new position back into the old one. This is what is meant when it is said that force is stored up in the grey matter of the nervous system and liberated at intervals. Such a statement means, that the highly complex structure of the molecules of the grey matter is built up by the action of force on the atoms; that the force so employed lies latent in the structure; that the structure so formed is an unstable structure; that the bonds of union between the component sub-molecules are frail and easily broken; that the frailest portions of the structure may be shaken off by a very slight force, and that, when parts are thus shaken down, the force employed in building them up is set free. It is set free in much the same way as the force stored up in a pile of bricks is set free when the bricks fall down, and waves of sound and heat are straightway projected in all directions.

Such an alteration of the structure of a molecule as is above described, since it is an alteration from a more complex to a more simple structure, is of the nature of a decomposition, but since the molecule contains just as many atoms after receiving the shock and undergoing the rearrangement as it did before, there is no decomposition in the chemical sense. When a molecule is decomposed in the chemical sense, the atoms of which it is made up are not merely rearranged within the molecule; they are torn completely asunder, and may be united with foreign atoms to form entirely different compounds. This is not the case in the process that has been described. What happens there is a rearrangement of atoms within the molecule without any disruption of, or loss of atoms by, the molecule. The chemical composition of the grey matter is unimpaired, and a mere lifting of the displaced atoms into their original places will completely restore the *status quo ante*. Thus it can be easily understood how the structure of the grey matter may be continually undergoing decomposition during its functional activity without being permanently or

materially impaired. It will be convenient to use the term "decompounding" for this form of rearrangement, reserving the term "decomposition" for the major process which involves destruction. The process of decompounding, with the liberation of force which necessarily accompanies it, together make up the nervous discharge.

Several accompaniments and consequences of this process require notice.

If we suppose, as the extreme complexity of the molecule and what is known of its constitution entitle us to suppose, that all the atoms and groups of atoms that occupy the position of top bricks are not equally unstable, but are bound to the main body of the molecule with different degrees of firmness, then it will be evident, that the number of atoms that are shaken out of their places on the impact of a shock will depend on the magnitude of the shock. A stronger shock will displace more atoms than a weaker shock; and since the greater the number of atoms displaced, the more force is liberated, it follows that, other things being equal, the *greater the disturbing force, the more powerful will be the resulting discharge.*

Next we have to notice, that the force set free by the decompounding of a molecule itself acts as a disturbing agent on the molecule. A very feeble impact may be able to displace only the most loosely attached atoms. But these atoms in their fall liberate force, and the force thus liberated may be enough to displace the atoms of the next degree of instability. The fall of those atoms may still further spread the process. Just as the fall of one brick on the top of a pile tends to knock down other bricks; just as the explosion of one barrel of gunpowder in a magazine tends to explode other barrels; so the fall of one atom of a molecule tends to knock other atoms out of their places. Hence *a discharge once begun tends to get stronger.* Again, the force set free by the decompounding of a molecule is not confined within the molecule, but diffuses around, much as the waves of sound and heat initiated by the fall of a pile of bricks spread around in all directions. But every molecule of grey matter is surrounded by other molecules. Hence the force set free

from one molecule must impinge upon others. And when a force impinges upon the molecules of the grey matter, it will, if strong enough, shake their atoms out of place. Hence the discharge of a molecule tends to cause the discharge of other molecules. In other words, *a discharge once begun tends to spread.*

If a number of molecules are aggregated together, and some of them are made to discharge, a certain amount of force is liberated—the discharge attains a certain strength. From the two preceding paragraphs it appears that an increase of strength may accrue to this discharge in two ways. It may spread to more molecules, and be augmented by the force liberated from them, or it may spread deeper into each molecule and be augmented by the fall of additional atoms in each discharging molecule without any increase in the number of molecules discharging. The former is an extension of the discharge—an increase in the area that discharges with a given intensity; the latter is an increase in the intensity of the discharge that issues from a given area. Hence we recognise, that *a discharge may be augmented either by extending in area or by an increase of intensity.*

The force set free by the decompounding of a single molecule is of course inappreciably small: but the molecules are indefinitely numerous, and the accumulation of an indefinitely large number of inappreciably small forces of similar character results in a force of appreciable magnitude.

We have seen how the force stored up in the grey matter is liberated; we have now to ask how it is replaced. By what process, under the operation of what causes, do the displaced atoms return into their less stable position on the molecule? The only answer that we can at present give to these questions is that we do not know. We know that as a matter of fact the atoms are replaced, or that others are substituted for them, for the nerve-centres continue to discharge and yet do not become decomposed. It is obvious that, if the discharge continued without restoration of the fallen atoms, the process could not go on long; for each successive decompounding would reduce the atoms to simpler and simpler combinations, until at length the molecule would be reduced from a complex

cluster of many groups of atoms and clusters of groups, to a disorderly heap of single particles. Long before this stage was reached, however, the molecule would split up into smaller molecules, and entirely new combinations would be formed. The grey matter would be chemically decomposed. We know, as a matter of fact, that no discharge is continuous. Every liberation of force is followed by an interval; and a prolonged discharge is made up of numerous separate waves of discharge separated by intervals of inactivity. It is fair to suppose, that during these intervals the fallen atoms are being replaced into a position of greater instability, from which they are again displaced as the discharge is renewed. This replacement of the fallen atoms requires time: it requires more time than is required for their displacement. Hence when discharge follows discharge rapidly and for a length of time, the process of displacement outstrips the process of restoration. The restoration of the fallen atoms being incomplete, the impinging force finds opposed to it a less number of top-bricks—of unstably placed atoms—in the molecule; and the topmost, or most unstably placed atoms being of course those whose restoration is the first to fail, the unstable atoms that are now exposed to the impinging force are less unstable than those which were at first exposed; they require a stronger impact to displace them. As the process continues, these atoms also fail to be replaced, and thus, as discharge is repeated, successive layers of atoms in the molecule are laid bare, each stratum being more stable than that which overlay it. At length a stage is reached at which the stability is too great to be overcome by the impact that is applied, and the discharge ceases. In this process we see the explanation of the occurrence of exhaustion and of the restorative potency of rest. After a long interval of rest so high a degree of instability is gained, that the impact of an infinitesimal force is enough to initiate a discharge, which then appears to be spontaneous, and which, once begun, is a cause of its own continuance. After a considerable discharge the degree of stability becomes such that stronger and stronger stimuli have to be applied in order to maintain the discharge. The stimulus of the voice, for instance, is replaced by that of the whip, and the whip is

supplemented by the spur. On this hypothesis we see too why a short period of very vigorous exertion is more exhausting than much greater exertion spread over a longer proportionate time. In the latter case the intervals are occupied with the process of rebuilding, and their aggregate is sufficiently large to allow of a higher standard of structure being maintained than in the former.

By what agency this rebuilding of the molecule takes place we do not at present completely understand, but we know that there are many similar processes continually going on in the bodies of organisms. The development of the body out of scattered material by the accretion of molecule to molecule and cell to cell is a process of precisely the same character, and one of whose immediate causes we can give no account. The whole function of nutrition, of which the restoration to integrity of discharged nerve-centres is but a single instance, is a process of the same nature. All activity of function is attended by oxidation—by the degradation, that is to say, of some portion of the active tissue into a simpler molecular structure, and all growth, development and nutrition is the integration or redintegration of a simpler into a more complex molecular structure. Since the recompounding of the grey matter after discharge is a portion of the general process of nutrition, it will as a rule vary as general nutrition varies. Hence we find that those people who most readily recoup themselves after exertion—those who require least rest and least sleep—have the reputation of being the longest lived. Hence we find also that such people are the least obnoxious to adverse influences—to extremes of heat and cold, to the influence of alcohol and other drugs—and most readily overcome the effects not only of fatigue, but of various excesses.

The longer the molecules are left undisturbed, the longer does the building-up process continue, and the more and more unstable is the position in which their peripheral atoms become arranged. When a considerable time has elapsed since a molecule was discharged, the position of the atoms becomes so extremely unstable, that the impact of an infinitesimal force will be enough to upset them. It will further follow, that when atoms are in this extremely unstable condition, not only

will they be upset by an extremely minute force, but that, once disturbed, the number of atoms that undergo rearrangement will be relatively great. Where a large number of atoms are in extremely unstable positions, a force which is sufficient to dislodge only the most unstable of all will, when it is reinforced by that liberated in the fall of these atoms, produce a widespread collapse; just as a house of cards falls into complete ruin when a single card is displaced. And the more atoms that are displaced, the more force is liberated, the more intense the discharge. Hence the longer a molecule has been left undisturbed, the more prone it is to discharge, and the more force does it liberate when the discharge occurs. In this elementary fact we see the explanation of the recuperation that takes place after rest, of the ease with which exertion of all kinds is undertaken in the morning, of the many phenomena of fatigue, exhaustion, &c. Here also is the explanation of the so-called spontaneous discharge, an occurrence which is never actually spontaneous, but which occurs when the molecules are fully charged, on the provocation of a force so small as to be insignificant. On the other hand, when the molecules have been frequently discharged and have had but few and short intervals for recuperation, the more unstable atoms will all be displaced; and as more and more stable strata are laid bare, greater and greater amounts of force are necessary to displace them; and when an atom is displaced, the connections of surrounding atoms will be so firm, that the displacement is limited to a small area, and the impact of even a considerable force will evoke but a very small discharge.

The function of the nervous system is, as we have seen, not only to store up and expend force, but to carry it from one part of the body to another—to deliver it from the store to whatever place it may happen to be wanted in. It remains to show how this transference is affected. It is scarcely necessary to say, that the liberated force exercises no choice as to the direction it shall take. It spreads from the point of liberation equally in all directions—provided all directions are equally permeable. But all directions never are equally permeable. Not only are the surrounding molecules, in so far as they are of the same nature, differently circumstanced, some being

older, some more recently discharged, some better situated for recuperation, than others, but every molecule possesses polarity such that it is more sensitive to shocks arriving in the direction of one of its axes, than to shocks arriving in any other direction, and delivers its discharge in greater intensity along this axis than in any other direction. Hence a discharge will spread more readily where the molecules have their poles parallel than where they are higgledy-piggledy.

The more unstable the molecule, the more completely it is discharged by an impinging force, and the more force it liberates in its discharge. Again, the stronger force is the more efficient disturber, and hence for two reasons the discharge will spread most along the direction in which the molecules are most unstable. Now suppose that the unstable molecules are packed inside a tube whose walls are constructed of molecules so stable that they will not discharge at all, and suppose that they are packed with their polar axes parallel. Manifestly a discharge initiated at any point in the tube will travel with great facility along the length of the tube.

Such is the structure of a nerve-fibre. It is composed of an axis-cylinder of grey matter, enclosed in a tube of substance that will not discharge. We have seen that each molecule that discharges adds something to the force of the total discharge, and hence, if the supposed view is correct, the nerve current ought to be stronger the longer the tract of fibre that it has traversed; and it is a well-established fact in physiology that "if the irritant remains the same, the longer the portion of nerve irritated, the stronger is its action on the muscle."

Having such a construction as described, when a medullated nerve-fibre transmits a wave of force, this wave is entirely confined to the axis-cylinder in which it runs from end to end. The stably arranged sheath prevents all diffusion of the discharge. But all nerve-fibres do not possess a medullary sheath. Very many fibres are naked throughout their length, and all are destitute of sheath at their ends. From this we may infer that some nerves allow, under some circumstances, a certain lateral diffusion of the currents passing along them, and that this diffusion may occur at the ends of all nerves. We need not suppose, that where there is no sheath there is

no restriction of this lateral diffusion, nor that where a nerve-fibre loses its sheath the currents spread from it uniformly in all directions; but we may be certain, from the existence of the sheath, that its absence allows to the currents some lateral diffusion. If not, there is no need for a sheath. When we look at a section of grey matter under the microscope, we see a number of fibres traversing in various directions an intervening substance. The fibres are not divided from the inter-fibrous substance by any sheaths or membranes. No conspicuous difference can be found between their substance and the substance in which they are imbedded. As a rule, the tissue must be considerably altered by immersion in different fluids, or by staining, or both, before any difference at all is discernible; and when the fibres are in this way made conspicuous, very many of them taper off to an extreme tenuity and become at length indistinguishable from the ground-substance. By more elaborate preparations, and by better methods of staining, the fibres may be tracked farther and farther; they may be traced into prolongations of greater and greater tenuity; but the more perfect our methods, and the more laborious our observations, the more certain it becomes, that the great majority of nerve-fibres taper off into fine points which lie imbedded in the ground-substance. From these observations three inferences may justifiably be drawn. From the different action of reagents upon the fibres and on the matrix, we may infer that the molecular constitution of the fibres is different from that of the matrix. From the precautions and preparations necessary to display this difference, we may infer that it is not very great. And from the increasing difficulty in tracing the fibres as they extend further from their main trunk, we may infer, that the difference between the molecular constitution of the fibres and that of the matrix diminishes as the former approach their free terminations. Viewing them with regard to their molecular stability, it can scarcely be doubted that the molecules of the fibres are more unstable than those of the matrix; but considering the similarity of the two substances, the difference of stability is probably not great, and becomes less as we approach the termination of the fibre. If we accept this view, in favour of which

there are several arguments that cannot be reproduced here, we are committed to certain other views regarding the transmission of waves of force, or discharges along these fibres. Suppose a molecule in one of these fibres to discharge. Force is liberated, and, if the molecules of the fibre are supposed symmetrical and similar, spreads in all directions both along and across the fibre. When the discharge has spread through the whole thickness of the fibre, so that all the molecules in a transverse section are discharging, it impinges against the molecules of the surrounding matrix, which, by hypothesis, are not so easily discharged as the molecules of the fibre itself. It is obvious that the next event will depend entirely on the strength of the discharge. If the impact of the force is strong enough to knock down, not only the loose atoms that compose the molecules of the fibre, but also the somewhat more firmly compacted atoms that compose the molecules of the matrix, then the molecules of the latter that abut upon the fibre will be discharged. Discharging, they will liberate force, and will pass on the movement in increased intensity to the molecules lying further from the fibre. Hence, powerful discharges will not only traverse the fibre, but spread into matrix also. If, however, the discharge passing along the fibre has not sufficient force to upset the more stable molecules of the matrix, it will remain limited to the fibre, and will pass along its length without becoming laterally diffused. To suppose that the waves of discharge in the nerve-centres are entirely limited to the fibres, is a mistake, and a very vital mistake. Whether they remain confined to the fibres, or whether they overflow and permeate the imbedding substance, depends, other things being equal, on the strength of the discharge; and the possibility of their escape from the limits of the fibre into and through the surrounding substance is one of the most important properties of nervous tissue, and the foundation of one of the most valuable of all faculties—the faculty of progress.

Not only may a current, if sufficiently powerful, escape from the fibre and spread through the ground-substance, but there are circumstances in which a current in a fibre, however feeble, *must* become diffused into the surrounding matrix. Suppose

the case of a fibre which tapers off and ends, after a longer or shorter course, as an attenuated point. What becomes of a current of force when it reaches this termination? Again, we must consider the process as an affair of decompounding molecules. If we take a number of cross-sections of the tapering nerve, the area of each section becomes less and less as we approach the point. The structure of the fibre being supposed uniform, it follows that the number of molecules exposed in each cross-section becomes less and less in each successive section. In other words, as the wave travels along the fibre, the discharge of a number of molecules is passed on to a less number. The force impinging on the less number is not less than the force impinging on the greater number. On the contrary, it is greater, for it is reinforced by the discharge of the latter. But the same amount of force when applied to a less number of molecules will produce a greater effect upon each than when applied to a greater number,—it will discharge the less number more completely. In other words, as the discharge approaches the termination of the fibre, it increases in intensity. When the discharge reaches the extreme point of the attenuated fibre it will have a considerably greater intensity than it had where the fibre was considerably thicker; that is to say, its impact against the molecules that lie round the point of the nerve will be more powerful than its impact against the molecules that bound the fibre laterally; and hence a discharge which is not powerful enough to escape laterally from the fibre, may yet have sufficient impetus to escape at its termination. When we remember that the molecules of the matrix, although more stable than those of the fibre, are not much more stable, it will be evident that when currents of various intensity are very frequently passing along the fibres, there must occasionally occur some that are not powerful enough to discharge those molecules of the matrix that surround the course of the fibre, but yet gather intensity enough to discharge those that surround its termination.

So far, we have discussed the nervous discharge without once mentioning the nerve-cells, an omission that may well arouse astonishment, but one that has been made designedly.

It is customary to regard the nerve-cells as unique bodies possessing wonderful and even miraculous powers. They are able, it is said, not only to transfer force from one fibre to another, but to transmute a wave of force into a conscious sensation, and an idea into a movement; they are able to suppress movements; not only force but memories are stored away in them; they are the active portion of the nervous system, the remainder being either mere packing or merely subsidiary and supplementary material. Most of these views are certainly erroneous, and all require modification. The nerve-cells cannot transmute a movement into a feeling or an idea into a movement. They cannot contain memories. They are not the only active portions of the grey matter. That they are active constituents of the grey matter admits of no doubt; but they are far from being the only or even the most important of such constituents.

The nerve-cells are continuous with the fibres. It is common to speak of a fibre as entering a cell; but the connection between them will be more correctly conceived if the cell is regarded as a bulging of the fibre, and the fibre as a prolongation of the cell. There are, it is true, cells which appear to be isolated, and which have no discernible connection with any fibre; and it is extremely significant that these cells have no cell-wall, and are less distinguishable from the matrix in which they are imbedded than any other form of cell; but the great majority of cells are continuous, with the substance of one or more fibres. Between the molecular structure of the cells and the molecular structure of the fibre there appears to be some difference, but assuredly the difference is not great. There is no line of division between them. The one merges by insensible degrees into the other. Their optical appearances and their response to reagents are almost identical, and whatever differences can be found to exist between them are largely explicable by the greater bulk and different form of the cell—by the different mode of aggregation of the molecules. Supposing, for the sake of argument, that the cells are composed of molecules precisely similar to those of the fibres, still the function of the cells, or the manner in which their molecules as a whole behave under the impact of a force,

will be different from that of the fibre-molecules. Let us see what this difference will be.

The main difference in aggregation between the cell and the fibre, is that within any given distance, greater than the diameter of the fibre and less than that of the cell, there is a much greater number of molecules in the cell than in the fibre. Supposing a cell to have a diameter only ten times as great as that of a fibre, which is a moderate average, and to be of similar uniform molecular constitution, then, if discharges of equal intensity are started from the middle point in the long axis of each and spread to the circumference, the discharge in the cell will liberate 1000 times as much force as that in the fibre, and will occupy only 10 times as much time, so that in each unit of time the cell will liberate on the average 100 times as much force as the fibre. In other words, the discharge in the cell is of a more explosive character than that in the fibre, supposing both to be similarly constituted and to be similarly disturbed by equal forces. It is possible that the molecules of the cell are more unstably constituted than those of the fibre, but of this we have no evidence. But since most cells are a meeting-point for several fibres, it is obvious that while the impinging force which sets up a discharge at any point of a fibre can arrive in one of only two directions, that which discharges a cell may arrive from several directions, in some cells probably from any direction.

Since a large cell contains more molecules than a small one, it will, if similarly constituted, liberate more force in its discharge; and since the more explosive character of the discharge in the cell depends on the longer radius that can be drawn from the starting-point of the discharge to the periphery of the cell, it is obvious that a large cell will liberate force in a more explosive manner than a small one. Hence where an effect depends on both the amount and the suddenness of the discharge, the larger cell will have the advantage in both ways. It is significant that large nerve-cells are specially characteristic of motor regions.

Reference has been made to the fact, that some cells have no fibres in direct connection with them. Such cells—I speak only of those that are admitted to be nerve-cells—must have

the same fundamental function as other nerve-cells—must accumulate force, and discharge it upon provocation. But since they have no fibres, the only possible way in which the jar necessary to upset the equilibrium of their molecules can reach them is through the matrix in which they are imbedded; and the only possible way in which the force liberated by the discharge of such cells can escape is through the matrix. Now we can see the significance of the fact already alluded to, that apolar cells are but indefinitely demarkated from the matrix in which they lie. Were it otherwise, they could exercise no function, for any boundary between the discharging molecules of the matrix and the charged molecules of the cell would prevent the former from acting on the latter. The same barrier would imprison the discharge and prevent it spreading. As it is, such cells may be looked on as but slightly differentiated portions of the matrix. They are separated from it by no defined boundary, and can freely receive and impart impulses from and to it. The existence of such cells is an additional argument for the active function which is here claimed for the matrix of the grey matter.

Having now considered separately in the fibre and in the cell the molecular movements which constitute their function from its physical aspect, let us see in what way these movements will be modified in their passage from the fibre to the cell and from the cell to the fibre. For the present purpose, those cells which are directly continuous with fibres may be regarded as bulgings in the course of fibres—as protuberances at a place of division. Now suppose a wave of discharge to proceed along a nerve-fibre and to arrive at one of these bulgings; what will happen? From the point at which the nerve joins the cell a discharge will spread through the whole cell-substance. The cell will discharge in an explosive manner, and will liberate a relatively large amount of force in a short time. This force, liberated by the molecules within the cell, seeks to escape. A head of pressure is established which tends to a restoration of equilibrium by flowing off in directions in which pressure is less. The force is pent up in the cell much as condensed gas is pent up in a soda-water bottle. It presses in all directions against its envelope. But

the boundary of the cell is composed of material which is, metaphorically speaking, too rigid to yield to the pressure. The bounding molecules are too stable to be upset by any force that can be liberated within the cell. The force has to escape wherever there are openings in the boundary—in other words, wherever the cell-substance is prolonged into fibres; and these openings are few and small in comparison with the whole area of the cell. As in other cases where a considerable pressure escapes through few small openings, the rush through each opening will be of considerable intensity—of much greater intensity than would be the case if the force escaped freely over the surface of the discharging cell. We see, therefore, that for many reasons the discharge of a cell along the fibres emerging from it will be far more powerful and far more intense than the discharge whose entrance set up the discharge of the cell. In other words, a discharge in passing through a cell undergoes three changes. 1. Except in bipolar cells, which are few, it is communicated from a single fibre to several fibres. 2. It is increased in force. 3. It is increased in intensity. This increase in the force and intensity of the discharge is very great and sudden, and in comparison with it the increase gained by a discharge in passing along a fibre is insignificant in amount and, what is perhaps of more importance, is gradual in its accession. For these reasons the term "discharge" is often limited to the process as it occurs in the cell; the process as it occurs in the fibre, although identical in nature, being neglected.

So far we have dealt with the physical functions of the grey matter as occurring in a structure of constant composition. We have considered the force set free by the discharge of the molecules as travelling from place to place, becoming diffused, impinging against other molecules, and effecting their discharge. We have followed the course of the discharge from the fibre to the cell and from the cell to the fibre, and we have not forgotten that it may permeate the matrix also. But every disturbance that has yet been noticed is purely and necessarily a temporary one. If the change produced in a molecule by the rearrangement of atoms which constitutes its discharge were a permanent change, it is obvious that the

molecule would be of no further use. Thenceforth it would be only an obstruction and an encumbrance. Only on condition that the fallen atoms are replaced, and the molecule restored to its previous condition, is a further exercise of function possible. All that we have at present considered is an oscillation of structure from a condition of less stability to a condition of greater stability and back again; this oscillation of structure being accompanied by a concomitant variation of function, the molecules now liberating, now accumulating force. We have now to notice that, besides this temporary effect, there is produced by each discharge a permanent effect, which is of enormous importance, and which is the basis of the psychological function of the grey matter, as the changes of which we have hitherto spoken are the basis of its physiological function.

Every wave of force that passes through the grey matter discharges all the molecules against which it breaks that are sufficiently unstable to be affected by it. Whether a molecule is discharged by a force, depends upon three conditions. First, on the constitution of the molecule as stable or unstable; second, on the degree to which, if unstable, it has recovered from a previous discharge; and third, on the direction with respect to its polar axis from which the force arrives. Some molecules, even of extreme complexity, such as those composing the medullary sheath of nerves, are of such stable constitution as to be incapable of discharge, and probably all molecules in the grey matter are not equally susceptible to this change. A molecule which has recently been deeply discharged and has not had time for recuperation, will not, it is manifest, suffer discharge except on the impact of a very powerful force. But supposing all the molecules of a tract of grey matter to have a similar constitution, and to be equally charged, then the facility with which a discharge passes through it will depend on the condition of the third factor—on the degree to which the direction of the wave approximates to the direction of the poles of the molecules. Suppose that the poles of all the molecules are parallel to one another, and suppose that the impinging force travels in the polar direction. Then it is obvious that the facility for the passage of the

discharge is at a maximum. Such a molecular structure is the most permeable possible. But now suppose that the discharge passes through a structure in which the majority of the molecules have their poles in the direction of the course of the discharge, but some are placed obliquely at various degrees of inclination to this direction. Such a structure is manifestly less permeable to the discharge than the previous structure. Hence, when discharges of equal strength enter a given thickness of each structure, a less amount of discharge will emerge from the second tract than from the first. A residue of force equal to the difference between the two discharges on their exit has been retained in the second tract. What has become of this force? In what way has it been disposed of? *It has been used up in shifting the molecules so as to bring their poles nearer to parallelism.* Whenever a discharge passes through a tract of grey matter which has not reached its maximum of permeability, a part only of the discharge issues from the tract, and the remainder is spent in rendering the tract more permeable. The less permeable the tract, the greater the proportion of the discharge used up in increasing its permeability. The more permeable the tract becomes, the less the proportion of the discharge devoted to this purpose, and the greater the proportion that emerges. When the permeability reaches its maximum, the discharge that emerges is actually greater than that which enters, owing to the accession that it receives from the decompounding of the molecules. How the passage of a wave of force through an aggregate of molecules of different degrees of stability changes the less unstable molecules into more unstable ones, is perhaps not yet precisely ascertained. Those who desire to pursue the subject will find it discussed at length in Mr. Herbert Spencer's 'Principles of Biology,' §§ 3 to 23, and 304; and in his 'Principles of Psychology,' §§ 223 to 230. It will be enough here to quote two illustrations from those great works, which serve to give an accurate notion, not indeed of the kind of change that takes place in this particular instance, but of the manner in which a force in passing from particle to particle may be expended in two ways, part being absorbed in altering the disposition of the particles, and the rest being passed on

to the extremity of the series of particles. "Take the rude analogy furnished by a row of bricks on end, which overthrow one another in succession. If such bricks on end have been adjusted so that their faces are all at right angles to the line of the series, the change will be propagated along them with the least hindrance; .or, under certain conditions, with the greatest multiplication of the original impulse. For when so placed, the impact each brick gives to the next, being exactly in the line of the series, will be wholly effective; but when they are otherwise placed it will not. If the bricks stand with their faces variously askew, each in falling will have a motion more or less diverging from the line of the series; and hence only a part of its momentum will impel the next in the required direction. . . . Suppose that the row of bricks, which were at first very much out of parallelism, have fallen, and that part of the motion given by each to the next has gone towards bringing their faces nearer to parallelism; and suppose that, without further changing the positions of their bases, the bricks are severally restored to their vertical attitudes; then it will happen that if the serial overthrow of them is repeated, the actions, though the same as before in their kinds, will not be the same as before in their degrees. Each brick, falling as it now does more in the line of the series, will deliver more of its momentum to the next; and less momentum will be taken up in moving the next towards parallelism with its neighbours."

A further consequence of this increasing permeability of the tissue to the discharge will be the increasing limitation of the discharge to narrower and narrower channels. "Along a line of discharge, there is a genesis of the matter most capable of communicating the discharge. Every time an incipient nerve is traversed by another wave of molecular motion, there is apt to be a further formation of the molecules which are isomerically transformed by the wave and pass it on in being transformed. This process acts with continually increasing power, for two reasons. One is that progressing limitation of the wave to a well-marked line, enables it to produce more decided effects along that line. An illustration will here help us. When a body of water flows over a surface

offering no distinct course, it thins out into wide-spread shallows near its margin, where it is almost motionless; and it has but little motion even along its central deepest parts. But if the inundation is long continued, the abrading action of the current along these central deepest parts where it moves fastest, tends to deepen its channel there more than elsewhere. A secondary result is a retreat of the water from the shallows —the current becomes more concentrated. In proportion as it becomes more concentrated, the force of its central part becomes greater still, and the deepening more rapid; which entails a further drawing-in of the margins and a further addition to the excavating force. So that the growing definiteness of the current brings a growing power of making its channel quite definite. Now though in the case before us we have not a motion of matter over matter, but a transfer of molecular motion from molecules to molecules, the parallel holds. Any greater effect produced by the transfer along one part of its originally broad course, similarly tends to concentrate the transfer along this part, and thus to intensify the action which makes this part a precisely marked channel. A further facilitation results from an absolute increase in the amount of the nervous discharge. The more permeable the line of molecules becomes, the greater becomes the initial quantity of molecular motion it draughts off. As with water, the formation of a definite channel not only makes the transfer easier and adds to the excavating power of the current, supposing its volume be constant, but also (if the reservoir can supply more) augments the volume carried away, which again adds to the excavating power; so the formation of a better line of nervous communication is followed by an increase of the wave that sets out to traverse it, and a consequent increase in the channel-making action. Once more, every addition to the molecular motion transmitted, adds to the effectiveness of each discharge in overcoming an obstacle. Suppose the greater part of its channel has become tolerably permeable, but that at some place in it the colloidal matter is less transformed than elsewhere into the fit type. Then the more the rest of its channel increases in permeability, the more powerful must be the wave of molecular motion brought to

bear on the untransformed part, and the greater must be the tendency to transform it. Hence the channel will progress towards a state of uniform permeability."

From what has gone before, the reader will have perceived that the freely permeable channels of communication, or the lines of aggregation, of the most unstable molecules are the nerve-fibres. This is common knowledge. What is not so generally recognised is that other channels of communication exist. Apart from the foregoing reasoning and from all question of the mode of origin of nerve-fibres, there is ample evidence of the existence of such other channels. It is a fact that a vast number—an overwhelming majority—of the nerve-fibres in the grey matter end at one extremity as fine points—have no discernible channel into which the waves of force that traverse them can be delivered. Each advance in our means of research enables us to trace these nerves a little further; but still their destination eludes us, the newest researches show us no more definite ending than a tapering point. If the matrix of the grey matter has no carrying function, each of these fibres is functionless. They are all histological abortions—tentative efforts, which have failed, to connect distant cells. An hypothesis which involves the supposition that the immense majority of nerve-fibres are so much waste material, is monstrous. According to the views that are here advocated, such fibres are merely portions of channels. They are those portions in which the process of mobilisation is complete; and they are supplemented by prolongations in which the process is incomplete—in which the molecules that have acquired the maximum of instability, and at the same time have acquired the power of acting differently on reagents and on light, do not constitute a sufficient proportion of the molecules forming the whole channel to give it, as a whole, optical and other qualities so distinct from the surrounding matrix as to enable us to recognise it as different. This is not mere hypothesis; for where we have a reagent which affects more powerfully than previous reagents these altered molecules, it enables us to trace the nerve-fibres further than a less powerful reagent. In other words, it brings into view prolongations of the fibres that were before invisible.

It is not, however, necessary to suppose that every nerve-fibre is prolonged by a definite, if invisible, channel to an ending in a nerve-cell. If the doctrines here advocated are correct, there must be many, very many fibres that have no such definite termination. It is one of the most vital principles of that neurology which forms the basis of psychology, that nerve-fibres and nerve-cells are not the only channels of communication in the grey matter. If the visible channels terminate in channels which are not sufficiently differentiated from the matrix to be visible, the inference is obvious that the discharge, which in passing along the visible channel is restricted to that channel, may, when it reaches the invisible channel, begin to spread into the matrix which is so little different. And if the fibres are gradually formed out of the matrix by the passage through it of discharges, then of course the matrix must be permeable to the discharge. This doctrine supplies functions at once to those apparently aimless fibres that, ending in free terminations, constitute so large a bulk of the grey matter, to the interfibrillar matrix which forms a still larger proportion of it, and to the apolar cells, which if visible fibres are the only channels of communication, are anomalous superfluities.

The conception of the physical condition of the grey matter that we have now reached is this. It is composed of complex molecules whose atomic constitution is such that they readily undergo internal changes without being disintegrated. These changes are of two main orders. One, which occurs on the impact of a force, consists in the fall of the atoms into more stable positions, and is accompanied by a liberation of force from the molecule. The other is the reverse change, and consists in the restoration of the fallen atoms to their previous positions, and is accompanied by a storage of force in the molecule. An illustration of these changes may be found in the waves of movement that pass over a cornfield under the pressure of the wind. Each stalk of wheat bends down under the impact of the wind into an inclined position, and in doing so strikes against other stalks and liberates the force which we recognise by rustling sound. No sooner is the bending complete, than the elasticity of the stalk asserts itself and the

ear rises into the erect position, ready for the next gust of wind to bend it down again. The two movements occur in alternation, one under the influence of an external force, the other under the operation of intrinsic forces when the outside force has ceased to act. The greater the impinging force, the greater the depression of the ear. The more stalks that are affected, the wider the wave; the more each stalk is bent, the deeper the wave. Finally the wave of movement passes from one end of the field to the other without any redistribution of the stalks of corn.

All the molecules of the grey matter have not the same susceptibility to these changes, or, what is more probable, the molecules are not equally susceptible in all positions with regard to the disturbing force. If we imagine the stalks of corn to be oval in section, it will be evident that they will bend more readily to a wind in the direction of the short axis of the oval than to one in the long axis. If there be much difference between the axes, the stalks will not bend at all in the direction of the long axis; and if each gust of wind which strikes them on either side of the long axis is able to twist them slightly, so as to turn them more broadside on to a similarly directed gust, these properties will have a still more extended analogy to those of the molecules of the grey matter.

Such being the structure of the grey matter, we have now to get a clear conception of its function in physical terms. To do so, we must imagine the structure thus described as permeated continually and throughout by innumerable waves of discharge. We must recognise that there is a circulation of force in the body, just as there is a circulation of matter. The latter takes place mainly in the blood-vessels, the former mainly in the nerves; and the two are in many respects comparable. Along every nerve-fibre gushes of force continually succeed one another, as waves of blood pass through the arteries. Every nerve-cell is, as it were, a heart which receives the current flowing into it, and discharges it with increased impetus. Every tract of matrix is comparable with the intercapillary tissue. As the blood plasma soaks through such tissue in no constant direction, without definite boundary

and with inconsiderable impetus, so the nervous discharge diffuses itself through the matrix of the grey matter in no definite channel, and with an impetus inferior to that which obtains in the nerves. As through the intercapillary tissue new capillaries are formed by protrusions from the old, so in the matrix of the grey matter new fibres are formed by protrusions from the old. Not only in the grey matter do these currents of force circulate; they are carried by the nerves to the uttermost parts of the body. There is scarcely a recess or nook of the organism to which they do not penetrate. While by far the greatest quantity is draughted into the muscles, large supplies go to all the tissues, where their access regulates those molecular disintegrations, rearrangements, and integrations that constitute nutrition and waste, secretion and excretion. The force thus expended is continually being renewed from the store that accumulates in the nerve-cells in the intervals of their activity. But however highly charged a nerve-cell may be, it never discharges spontaneously. However unstable its molecules, some force, even if infinitesimal, is needed to set the process going. Spontaneous movement, in the sense of movement originating without previous movement, is unknown—is, as far as we know the constitution of the universe, impossible. It implies a creation. We have to account, then, for the first shock that sets all the movement going. If we trace a discharge back from fibre to cell and from cell to fibre, we must always come at last to currents that arrive from outside the grey matter along the nerves that are called sensory or afferent. There is no other possible source for the first shake that sets all the movement going. And when we trace this afferent fibre to its extremity, we always find that it rises somewhere on the physiological surface of the organism—somewhere where forces from the outside world can act upon it, and originate the currents which, when they arrive at the grey matter, act as exploders. Further, we find that from all parts of the surface of the body nerve-fibres start and travel inwards to the great central masses of grey matter. And, furthermore, we find that several parts of the surface are especially modified, so as to collect and transmit to these nerves extremely slight disturbances arriving at the surface; such are the organs of

special sense. To complete our conception of the physical function of the nervous system, we must therefore picture to ourselves waves of force starting from all parts of the surface of the organism, and converging towards the great central masses of grey matter. Here they are sparse and feeble, there they are stronger and more numerous; and from special areas, such as the retina and the internal ear, they run in powerful currents. Arriving at the grey matter, these currents join in and reinforce the general molecular turmoil which goes on there; and which eventuates in far more powerful currents that are continually distributed to the rest of the organism. The greater part of these outgoing currents are supplied to the muscles, and are supplied, as has been said, continually. So long as the muscles are at rest, the currents reach them in sufficient amount to maintain only that incipient contraction which we call their tone; but that even this is continually varying in amount is shown by the success of the muscle-readers, who can discriminate varying shades of muscular tension which are unknown to the individual in whom they occur. From time to time stronger draughts of force issuing from the grey matter reach the muscles in sufficient quantity to initiate the contractions by which our movements are made. Thus, partly as mechanical movement, partly as heat and other forms of molecular motion, the force accumulated in the nerve-cells passes out of the organism, and the redistribution, so far as the organism is concerned, is complete.

Clinical Cases.

ABSCESS IN CEREBELLUM: SYPHILITIC SYMPTOMS: SUDDEN BLINDNESS: GREAT OCCIPITAL PAIN: GREAT BENEFIT FROM CALOMEL: DEATH: AUTOPSY.

REPORTED BY MONTAGU HANDFIELD-JONES, M.B. (LOND.), M.R.C.P.

G. R., æt. 46, coachman, admitted to St. Mary's Hospital under the care of Dr. Handfield-Jones on December 27th, 1883, suffering with severe pain in the back of the head and neck, accompanied by retraction of the head.

Patient is a healthy-looking, well-nourished man; is married and has thirteen children, all healthy; his father died of heart disease, his mother of uterine hæmorrhage at age of 54, brothers and sisters alive and well. Was always strong and healthy till four years ago, when he was attacked with rheumatic fever, which confined him to his bed for four months. Following the rheumatic fever, patient was seized with paralysis of right side of body; this, however, passed away entirely, but two years ago an attack of total blindness, accompanied by mental aberration, came on suddenly; for this he was eight weeks in Maidstone Hospital. The total blindness lasted about two weeks, and then sight was gradually fully restored. Syphilis is positively denied, but there are two places on lower part of left leg which look like the scars of rupial sores, and higher up on tibia of same leg is a large node; over crest of the left ulna is another node. There is a scar on the centre of the sternum, which has existed for a long while; patient says he had a sore there which came by itself, and after some time discharged a small piece of bone.

Patient is very deaf, and has been so for many years; he talks apparently quite rationally, but his own account of his illness is not trustworthy. According to his wife's statement, he has suffered for at least four months with sickness and pain in the back of the head on rising from bed in the morning; during the last three or four weeks there has been severe pain in the back of the head and neck. The pain comes on in fits,

which last two or three minutes, and are accompanied with retraction of the head. On December 24th, while at home, patient was very noisy and delirious; lately the attacks of pain have become much more frequent, recurring every half-hour or so. During the past week he has suffered with constant sickness; the bowels have not been open since December 19th, but he says he often goes for a fortnight without passing any motion, and for two days without voiding urine. There is no evidence of any accumulation in bowels or bladder; but patient has a stricture, which will not admit a No. 6 catheter. Heart, lungs, and other organs apparently quite healthy. Well-marked optic neuritis in both eyes; pulse 74, forcible, regular, not easily compressed; tongue coated, is protruded slightly to right; temp. 98·2; urine, straw colour, acid, sp. gr. 1035, no sugar or albumen, deposits crystals of oxalate of lime, contains some mucus and epithelium, total amount in twenty-four hours scanty. ℞. Calomel gr. v statim; leeches (12) to back of head; pot. iod. gr. x + pot. bromid. gr. xx + aq. ℥j, t. s. d.

Dec. 28th.—Patient passed a fair night, but the pain in the head was very bad at times; the attacks of pain occur rather less frequently. Vomiting took place after breakfast. Temp. this morning, 97·5. There was free bleeding from the leech-bites last night. ℞. Pil. hyd. c. col. gr. x statim; simple diet; beef jelly, O j; milk, O ij; ol. crotonis, ♏j + Ol. ricini, ℥ss.; ℥ij to be taken at a time.

Dec. 29th.—Patient is fairly rational, complains of great pain in the back of the head, says he feels worse, but the attacks of pain are not nearly so frequent. The bowels have been open after two doses of croton oil and an enema. Pulse 84; temp. 97·5. Blister (3 × 3) to back of neck.

Jan. 3rd.—Had severe pain on night of 31st, but very little since; has been taking potass. iodid. in gr. xx doses, also calomel in gr. j doses three times a day since December 31st. Is sufficiently rational now to help himself to food, but passes his evacuations under him, and seems to have no recollection of it. Shows his tongue and does what he is told. Pulse 93, fair force; regular, constant, and seems to have power over his limbs; no paralysis. Repeat blister to back of neck.

Jan. 5th.—Is much more rational, passed a very good night, is able to open his locker and get at food he had left. An enema was given to-day with much ado, patient struggling violently and shouting murder, thieves, &c. A few hours later he protested that he had not shouted at all. Still passes all his evacuations in bed. Pulse 108, soft, regular, no ptyalism.

Jan. 8th.—Has more pain to-day in back of head and neck. Retraction of head more marked; evacuations passed in bed; patient less conscious. Leeches (12) to back of head; calomel gr. j, quater die; potass. iodid. gr. xxv + pot. bromid. gr. xxv + aq. camph. ℥j, quater die. Temp. last night, 97·8; this morning, 97·5. Pulse 80, full and forcible.

Jan. 10th.—Spasms of the neck are very marked; arching of the neck is very distinct. Patient not more conscious; was awake all night, but is dozing now. Rep. mist. c. potass. iodid. gr. xl. Calomel, gr. j, semel die.

Jan. 21st.—Patient has been passing quieter nights since last report, but is still very stupid and troublesome at times. The arching back of the head is still well marked. Pulse 58, interrupted by a pause about every eight or nine beats. Temperature this morning, 97. Since the 14th inst. he has taken ol. morrh. ℥j, semel die; yesterday he was ordered calomel gr. iij, ter die; pot. iodid. gr. vj + aq. ℥j, omni hora.

Jan. 25th.—Not much change since last report. Constipation has been troublesome, but has yielded to ½ ♏ doses of croton oil. At 4 A.M. this morning, patient was suddenly taken worse. Æther and ammonia were administered every quarter hour, and a sinapism applied over the heart. He has rallied and is now sleeping. The iodide mixture has been stopped.

Jan. 28th.—Patient much more rational. Gets up of his own accord to pass his motions. Recognises his wife and child. His wife notices how much more clear he seems in his head. ℞. Ammon. carb. gr. v + tinct. cinch. co. ℥j, + infus. cinchon. ℥j, quater die.

Feb. 1st.—Yesterday was suffering severe pain at back of head and neck, the latter was much arched back; pulse about 80, very irregular; was ordered calomel gr. ½ duabus horis. To-day seems quite free from pain, and the head is not drawn back. The bowels are open naturally and he gets up to evacuate. Patient has his spectacles on and is trying to read his newspaper, but does not seem to make much sense of it. Pulse 93, soft, regular.

Feb. 14th.—No pain whatever in head. Wishes to go out. Moves head freely in all directions. Gums touched by mercury. Reads newspaper fairly well, so as to be intelligible to a bystander. Mental faculties quite clear now, memory returning. Left pupil noticed to be rather larger than right. Pil. calomel semel die.

Feb. 18th.—He has been steadily improving since the last report, and gets stronger every day. Says he feels better than at any time since admission; his memory has quite

returned, and his mental faculties seem perfect. Gets up at 6 A.M., and remains up all day; he walks somewhat feebly, but there is no entangling of feet or anything specially noticeable in his gait. Pulse 88, good force, regular. Temp. last night 98·6, this morning 98·8. Has suffered since last report with an attack of diarrhœa, but the bowels are acting well now. Both eyes to ophthalmoscope show fundus blurred and hazy; no disc can be defined, and only a feeble, whitish, irregular patch; retinal trunks look large and hazy, small vessels not seen. He reads fairly well with glasses.

Feb. 22nd.—Patient has been failing since the 20th inst. Has complained of much frontal pain and suffered much with diarrhœa. Deafness rather increased. Undue fulness of frontal veins noticed. Yesterday it was observed that he seemed to have lost control over the sphincter ani, the bowels seem to act before he is aware of it. Last night he suffered much with pain at the back of the head; this has now passed off; the attacks of spasm and arching back of the head have returned, together with more or less of all the old bad symptoms. The diarrhœa is still troublesome, but only in the early part of the morning. Pulse 84, full, regular. Temp. last night, 98, this morning, 97·6. Urine passed involuntarily into bed during night. Has returned to the calomel gr. ½ ter die.

Feb. 23rd.—Much pain over the top of the head; headache more frontal now than at back. Pulse at 3 P.M., 63. Temp. 98. ℞. Calomel gr. j, duabus horis. Patient was seized with sickness at dinner-time; vomiting continued on and off till evening. Death at 7.20 P.M. apparently from cardiac failure.

Post-mortem examination on Feb. 25th.—On opening the skull, the convolutions on the convexity of the brain are seen to be much flattened; the sulci are obliterated, the superficial veins engorged. On attempting to remove the brain the right half of the cerebellum was found firmly adherent to the dura mater of the anterior portion of the posterior fossa, so that the dura mater was taken away with the cerebellum. Much fluid escaped from the rupture in the floor of the third ventricle produced during removal of the brain. The right lobe of the cerebellum, contained two abscess cavities lined by a pyogenic membrane, evidently not of very recent date. The cavities were close beneath the surface (rather nearer the upper than the under part) of the cerebellum, separated from each other by a septum ½ inch thick of tough brain-substance; both contained thick green pus. The dura mater corresponding to site of abscesses was adherent to the cerebellum over a space equal to the size of a crown piece, and was much thickened by tough, translucent-looking material, probably gummatous in

origin and of the same nature as the morbid growth to be afterwards described on the periosteum of the tibia and ulna. The pia mater covering the cerebellum was thickened, especially near the site of the abscesses so probably blocking the foramen of Majendie and leading to distension of the ventricular system. In both eyes there was well-marked papillitis, though this was more apparent in right than in left; numerous small hæmorrhages were also well seen. The lungs, liver, spleen and kidneys were all intensely congested.

In connection with the periosteum of the left tibia there was a gumma equal in size to a walnut; on section, the centre was seen to be almost diffluent, while the outer firmer part was composed of grey, translucent tissue of a tough consistence. A gummatous growth of a similar nature was found in connection with the upper third of the posterior surface of the left ulna.

The morbid anatomy and pathology of the case recorded above are written in plain and distinct characters. In spite of his assertions to the contrary, it is clear that the patient had contracted syphilis at some period of his life; the scar on the sternum from which dead bone had been discharged, the gummata on ulna and tibia, the deposit in the dura mater over the site of the abscesses, are all so many definite and undeniable proofs. Again, it is obvious that the abscesses in the right lobe of the cerebellum were due to retrograde changes taking place in a mass of gummatous deposit, also that the distension of the ventricles causing flattening of the convolutions and obliteration of the sulci was caused by an accumulation of interventricular fluid, the escape of which was impeded by the pressure of the pia mater, thickened by inflammatory deposit, on the foramen of Majendie. Leaving, however, these clear and certain facts, we cannot fail to be struck by certain points in the history of the case, points which though of great interest in the clinical history, are yet by no means easy of interpretation:—thus, from what period in the history of the patient are we to date the onset of troubles due to specific cause? why with such a well-marked tumour were so many orthodox symptoms of cerebellar disease absent? can we fix the date of the onset of suppuration in the cerebellum, and can we account for the rapid return of all the bad symptoms, followed by death, when the patient seemed to all intents and purposes well out of the wood? The history given shows that the man was strong, able-bodied, the father of a large family, and in perfect health till four years ago; then comes an attack of rheumatic fever which confines him to his bed for four months, and following the rheumatic fever is an attack of right hemiplegia; now was this attack of rheumatic fever genuine

and true, followed by a hemiplegia due maybe to cerebral embolism, or was syphilis once more proving itself the great imitator, and during those four months of mimicry laying down an intracranial neoplasm, which by its inhibitory effect on the brain functions should produce the right-sided paralysis recorded? The history of this period is unfortunately faulty, and we have no report of the progress of the disease or treatment adopted, nothing but the bare fact that he got to all appearances perfectly well, and remained so until two years ago. One point we may call attention to, viz. the heart was carefully examined while the patient remained in the hospital, and at no time was anything abnormal noticed in the sounds; this fact alone militates against the idea of a rheumatic fever of four months' duration followed by hemiplegia presumably of embolic origin.

The next stage in the history of the case dates back to two years ago, when an attack of sudden blindness, lasting two weeks, and accompanied by mental aberration (extending over two months), is noted. This loss of vision is interesting in connection with the papillitis, which was demonstrated during life and at the post-mortem; if Schweller's view as advocated by Dr. Hughlings-Jackson be accepted, it is highly probable that this early and brief loss of sight was due to a reflex inhibitory influence exerted by some fresh activity or change in the growth or relations of the cerebellar tumour. Such an explanation is in no way incompatible with the idea, first put forward by Manz, that dropsy of the intersheath space of the optic nerve was the cause of the optic neuritis recognised during the patient's last illness. The inhibitory influence exercised at the early period would be lost, as soon as the brain tissue adapted itself to its new relations with the tumour, or maybe as soon as certain active changes in the life of the tumour ceased, while the influence of the increased intracranial pressure, demonstrated post-mortem, would have a lasting influence on the nutrition of the optic nerves. The presence of mental aberration is probably to be accounted for in the same way by a temporary inhibitory influence brought to bear on the higher cerebral functions: that the action of specific treatment may have had an important bearing on the duration of the blindness and mental derangement is of course to be borne in mind.

Coming now to the third or last stage in the patient's clinical history, we find that during the four months preceding admission to the hospital the patient had been steadily going down hill. His wife reports, that on rising in the morning at 6 A.M. he would go down to the stables to groom his horses, clean his harness, &c., but after some half-

hour's work would become so faint and dizzy, and have so much pain in his head, that he would be obliged to give in until after breakfast, when he would revive and be able to go out driving. By degrees this early morning faintness and pain in the head grew worse, and on rising from bed she had to give him strong beef-tea and a cup of strong coffee before he could start work; then gradually the pain in the head, especially at the back, became more continuous, till he experienced it all day long, and had at times to come home and lie down. The last three weeks before admission there was severe pain at the back of the head and neck, the pain coming on in fits, lasting two or three minutes, and accompanied by retraction of the head; towards the end of this time, i.e. shortly before his admission to the hospital, the higher cerebral functions were again attacked, and delirium with loss of memory is recorded. From this period till the date of death, the patient was under medical observation, and we have his symptoms accurately recorded. Some of these symptoms are such as we should clearly expect to be present, such as the constant vomiting, the pain in the head, the retraction of the neck, the constipation, the optic neuritis; but there are others which, in the light of post-mortem knowledge, we might equally have expected would not have been conspicuous by their absence. Thus at no time do we find the ataxic walk, the feet becoming entangled one with the other; no irregular movements of the eyeballs are recorded; there is no observation made as to the existence of tonic convulsions. Regarding the two latter symptoms, there does not seem to be any good recognised reason why they should be present in some cases of cerebellar disease and absent in others; but as regards the ataxic walk, I find that in a series of fourteen cases, notes of which I have by me, in only two instances was this symptom present, and in both these patients the mischief was very extensive and involved the central parts of the organ. With respect to the abscess in the cerebellum, the condition of the pus, thick, green and curdy, showed plainly that suppuration had occurred some considerable time previously; and as the temperature during his stay in the hospital was throughout subnormal, and none of the usual signs of suppuration setting in ever manifested themselves, it is highly probable that the purulent degeneration had taken place previous to admission. The extremely diffluent condition of the central part of the gumma on the tibia showed how ready the new tissue was to degenerate into pus; and granted a similar condition in the central part of the brain deposit, it is quite possible that the degenerative condition may have gone on so gradually that severe constitu-

tional disturbance was at no time excited. Nothing could have been more marked than the steady improvement which took place from February 1st to the 20th of the same month, and it may reasonably be suggested that the exhibition of small but continued doses of calomel played an important part in bringing about so very marked a gain, especially as all the bad symptoms returned on the drug being gradually dropped. It is plain, however, that the attack of cardiac syncope on the afternoon of January 25th showed on what precarious tenure life was held, and it was probably to some similar inhibitory influence brought to bear on the heart, that the death of the patient on February 23rd was finally due.

A CASE OF IDIOCY WITH UNIVERSAL RIGIDITY, THE RESULT OF SYPHILITIC DISEASE OF THE CENTRAL NERVOUS SYSTEM.

BY ANGEL MONEY, M.D., M.R.C.P.,

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A. R. H. a female child, aged 3½ years, was admitted into the Hospital for Sick Children, under the care of Dr. Dickinson, on April 3, 1883. She was an illegitimate child; her mother died of consumption, aged 31. The child had been bottle-fed all through. She was in good health till two years old, when she had two "fits" (? bilateral) in one afternoon, which were attributed to the teething. Three days after these fits, the child is said to have become unconscious. Since then she had not recognised friends, had not spoken, nor walked; though before the fits she was intelligent, could speak and had walked well. The bowels had been constipated. There was no vomiting.

On admission, the gums were spongy and swollen; the bowels were freely opened after a purgative; the patient vomited three times without apparent cause; the temperature was 104 at night, and 101 in the morning; the corneæ were clear; the discoloured teeth were ground down level with the gums in the upper jaw, and projected a little beyond the gums in the lower jaw. The upper part of the right external ear stood away from the side of the head. Unless the optic discs were too pale, ophthalmoscopic examination revealed nothing abnormal. The eyeballs were moved about thoroughly in every direction; they had no characteristic position; there was no facial palsy in crying; the tongue seemed to move naturally and equally. There was practically no intellect. The child seemed to observe a bright lamp, cried when food was put into the mouth; deglutition was natural. The mouth was very large, the face seemed ill-developed; the head was brachycephalic and small; its circumference measured 18½ in.; the measurement from ear to ear over the cranial vault was 12½ in. The legs, arms and trunk, when left alone and at rest,

were not stiff, or but very little so; the slightest disturbance however (sometimes the same thing seemed to occur apparently spontaneously) developed a marked tonic spasmodic condition of all the limbs, with opisthotonos of all parts of the spine, cervical, dorsal, but especially lumbar. The legs were straight at all the joints. The arms were rotated in and adducted at the shoulder; were extended at the elbow, but not fully; pronated in the forearm; the thumb was turned into the palm and the fingers flexed thereon, the wrists were semi-flexed. This posture of parts was constant, fixation occurring when spasm came on; the onset did not recall a spasm the result of strychnia poisoning or tetanus, principally in the want of suddenness, but perhaps there might be less difference between them than was supposed. The knee-phenomenon could be got on each side without difficulty; the ankle clonus could not be obtained, on account of the rigidity.

April 13th.—The patient was in much the same state, but the gums were practically well. The temperature had been below the normal since April 8th.

April 16th.—The patient swallowed badly, and sometimes did not attempt to swallow at all. The food regurgitated through the nose. Vomiting had been frequent.

April 17th.—The patient swallowed better in the night, but this morning she was worse again, and a muco-purulent discharge was flowing from the mouth constantly; there was no cough nor vomiting; the muco-pus bubbled up in the mouth. The bowels were constipated; a nutrient enema was given, which returned in half an hour. The voice became very weak last evening, so that she had not screamed when disturbed; this was a new feature. The back and neck were much more arched.

April 18th.—Excessive pallor appeared at 6 A.M., and was universal and ghastly. There was marked collapse. The patient was pulseless, the surface cold, the breathing shallow and actually ceased at times; the lips were bluish. The rigidity had more or less disappeared when the patient was disturbed. The hands were clenched, but could be undone; the fingers were flexed, the thumbs turned, the arms adducted, the elbows gently flexed, the forearms over-pronated; the legs were extended at all the joints; there was conjugate deviation of both eyes to the right. Death took place at 7 P.M.

Autopsy.—The weight of the body was 17½ lbs. The limbs were stiffened in the posture described during life. The cranium was asymmetrical, the left fronto-temporal region sloping, and not so round and full as the right. The frontals and parietals were 7 mm. thick at the thickest parts; the diploë was dense; the fontanelles were closed, and the sutures were united, but

there were no bosses. The dura mater was decidedly thickened, especially about the walls of the sinuses; there was nothing abnormal in the venous sinuses; there was much œdema of the subarachnoid space; irregular opacity and thickening of the pia mater were observed chiefly over the sulci; the pacchionian bodies were fairly marked; the brain was atrophied, especially its left hemisphere; its walls in the lateral and posterior parts were quite thin; the convolutions for the most part were picked out and well defined; many of them had a yellowish hue and were quite tough. The walls of the basilar artery were opaque white, thickened, and contained red tough thrombus. Nearly all the pia-matral vessels were thrombosed and their walls diseased. Nearly all the large arteries had thickened and opaque walls; long lengths of vessels were thus diseased; the whole of the basilar and the terminations of the vertebrals were very conspicuously diseased, and by far the greatest part of the length of the vessels forming the circle of Willis was diseased. The orbital plates crushed rather than fractured with the chisel, their diploë was thick and vascular. There was nothing abnormal in the choroids or optic discs. The orbital fat on both sides was dotted with points of hæmorrhage. The middle ears contained muco-pus, but the membranes were not perforated.

The pons Varolii was very hard. The cerebellum was perhaps a little tougher than natural; each half was equal, and of about the same size and shape. The medulla and part of the cervical cord stand out like a stick, before being put into spirit. All the spinal cord felt hard; on section, the anterior horns in places looked puckered and indented.

The heart weighed 2 oz., was healthy, as was also the pericardium. The larynx had a slight shaggy appearance, and red specks were dotted about, but nothing more definite was seen.

The left lung was adherent nearly all over, its weight was $3\frac{3}{4}$ oz. An area of consolidation, of ill-defined outline, of a dark dirty-red colour mottled with white, and of diminished consistence, was situated in the middle of the left upper lobe.

The right lung weighed $4\frac{1}{4}$ oz.; the upper posterior part of the right lower lobe, and the back half of the upper lobe were consolidated, of dirty dark-red colour, mottled with white, of ill-defined outline, and of diminished consistence. The bronchial tubes were all dilated, in one place having the finger-glove look; there was much mucus in the tubes. In the large intestines only the first six inches showed the lymphoid structures to be of a yellow tint, of a circular or oval well-defined outline, with a central dimple. None measured more than $3\frac{1}{2}$ millimetres across, many were smaller.

The liver weighed 12 oz., was of a dark red colour, and of

diminished consistence; there were no gummata. The pancreas was healthy. The spleen was big, of a dark red colour, weighed 2 oz., its consistence was not increased. The bronchial glands were enlarged; there were no cheesy glands anywhere. The kidneys weighed 2½ oz. together. The bladder was healthy. The muscles appeared to be a little pale.

Microscopical Examination.—Sections of the spinal cord show a diffuse form of sclerosis throughout the whole of its length. The cerebral substance has not been microscopically examined, but it may be assumed that extensive sclerosis is present in those convolutions which felt hard at the autopsy. Many sections made from the basilar and middle cerebral arteries exhibit well-marked arteritis, chiefly of the inner coat; in some sections the chief thickening is between the corrugate elastic lamina and the outer coat. Complete microscopical examination has not been made, owing to the fact that the specimen was kept in spirit to preserve its naked-eye appearances, and the spirit has now so altered the texture of the brain and cord as to render a complete examination impossible.

Remarks.—The case is of interest, as an example of the production of idiocy by coarse syphilitic lesions of the brain. In my own experience, which is limited to four post-mortem examinations of children idiotic from or soon after birth, some gross organic lesion has been present, and was in all probability the cause of the amentia. In one case of microcephalus I assisted at the autopsy with Dr. T. Barlow, and the cerebral hemispheres were, roughly speaking, converted into thin walled cysts over the greater part of their extent. In my second case there was a tumour, apparently gummatous, the size of a Tanjerine orange in the posterior region of the right hemisphere. The third example was one in which there was great atrophy of the right hemisphere, which was probably due to hæmorrhage, the result of instrumental delivery.[1] The last instance is the one here recorded. All the specimens are in the museum of the Hospital for Sick Children, Great Ormond Street.

My clinical experience at the hospital last mentioned leads me to believe that mental changes are by no means uncommonly produced by hereditary syphilis. I have met with children in whom the evidences of past syphilis were unmistakable, and in whom the mental condition was obviously not up to the average. This subject has been drawn attention to by an excellent contribution to 'BRAIN,' from the hand of Dr. Judson Sykes Bury, of Manchester.

[1] 'Transactions of Clinical Society,' 1883.

Reviews and Notices of Books.

A Treatise on the Chemical Constitution of the Brain. By J. L. Thudichum, M.D.

This work treats, in the first instance, of brain matter as a whole; several of its constituents are next studied, and an attempt is then made to obtain an insight into its normal and abnormal chemical functions.

The abundance of water in the neuroplasm is the first striking fact that meets the inquirer, and this, in conjunction with the peculiar manner in which the water is combined, engenders a great mobility of the ultimate particles.

The brain contains a considerable amount of an albuminous base. The bearing of this albumen, the author states, is governed by the components peculiar to the brain, which also by their manifold chemical affinities assist prominently in producing its singular mechanical arrangements, its chemical function, and its sensory and volitional action and reaction. These components he arranges in three groups: the members of the first contain five or six elements, one of which is phosphorus, and they are therefore termed *phosphorised bodies;* the members of the second group contain four elements, amongst them nitrogen, but no phosphorus, and are therefore termed *nitrogenised bodies;* while the members of the third group contain only carbon, hydrogen, and oxygen, and may be termed *oxygenised bodies.*

The phosphorised bodies the author names *phosphatides*, on account of their similarity to ordinary phosphates, and of these he gives several subgroups and species, of which lecithins, kephalins, and myelins are examples. These phosphatides Dr. Thudichum considers to be the "centre, life, and chemical soul of all bioplasm whatsoever, that of plants as well as animals;" and amongst their physical properties none, he thinks, are more deserving of further inquiry than those he describes as their powers of *colloidation*, without which "no brain as an organ would be possible, as indeed the existence of all bioplasm is dependent on the colloid state." When this hydric colloidation is at a maximum, the tendencies to decomposition seem to be at a minimum. These phosphorised bodies are readily able to combine with acids, alkalies or salts: but all their affinities, whether acid, alkaline, or alkaloidal, are overcome by water in quantity; the affinities for water, however, are overcome in turn by some metallic oxides, such as those of

lead, copper, manganese, and iron. The diversity of affinities is therefore very great, their exercise being greatly influenced by the mass of reagent and the mass of water present, and their interchange, accordingly, may produce an incalculable number of states of the phosphorised principles, and consequently of brain matter. From which it follows that neuroplasm, so far at least as the phosphorised principles are concerned, must yield obedience to even the slightest external chemical influence reaching it by way of the blood, taking up metals, acids, alkalies, salts, and alkaloids when so presented.

As to the colloid state above referred to, the author believes that its destruction in myoplasm, produced by the direct influence of the disease germ, leads to the fall of temperature in the collapse stage of cholera. Some bacteria possess the power of liquefying colloids, and the phosphatides may be capable of being similarly affected. The so-called softening of the brain consists, in the first place, merely in the loss of the colloid state by a smaller or larger portion of the nerve marrow. Such a softening may be effected by the bacilli of tubercle. After fluidification comes patholysis, the splitting up of the liberated immediate principles into their proximate nuclei. The two processes as seen, one in cholera and the other in softening of the brain, illustrate the acute and chronic forms of loss of colloidation and of patholysis under the influence of microzymes.

The *nitrogenised non-phosphorised* substances of the brain imitate in some respects the properties of the phosphorised, but as compared with them, their chemical characteristic is slight solubility; they are all firm compounds, and do not easily oxidise or decompose. Six groups are included under this head, of which phrenosin, psychosin, kerasin, and bregenin are examples.

The group of *oxygenised non-nitrogenised* principles consists mainly of alcohols and organic acids.

The brain, on the whole, is an aggregated mass of bioplasm, deriving its peculiarity mainly from specific chemical additions. The stroma of this bioplasm is made up of albuminous substances. Among these are small quantities of soluble albumen, partly exhibiting the properties of serum albumin; small quanties of fibrin, and considerable quantities of a globulin-like body, to which the author applies the term neuroplastin.

Into the methods of research pursued by Dr. Thudichum we shall not enter, further than to mention that by means of them he separates the brain into what he terms:

1. *First extractives.*
2. *Insoluble albuminous residue* containing plastin, albumen, and collagen, &c.
3. *White matter* obtained from alcoholic extracts, containing kephalins, myelins, cerebrins, cholesterin, &c.
4. *Buttery matter* by partial concentration and cooling of the alcoholic filtrate from the white matter, containing kephaloidin, paramyelin, more lecithin, &c.
5. *Last oily*, obtained by further distillation of the alcoholic

filtrate from the buttery matter, and containing lecithin and paramyelin, &c.

6. *Ultimate watery extracts* containing alkaloids, salts, &c.

To the mode of separation, the reactions, and the formulæ of many of the phosphatides the author devotes a large portion of his book. The constitutional formula, for example, of the principle kephalin is given as

$$C_3H_5\begin{Bmatrix} C_{17}H_{28}O_2\ (\text{Kephalyl}) \\ C_{18}H_{35}O_2\ (\text{Stearyl} \\ HO(OPO)\ C_5H_{12}NO \end{Bmatrix} = C_{43}H_{81}NPO_9$$

The conclusions to be drawn from the chemolyses of these phosphatides are thus stated. No glycero-phosphoric acid was obtained in any of them, and therefore glycerol cannot be essential to the composition of these bodies. Their only common feature was the presence of phosphorus, which by ordinary analytical processes was removed in the form of phosphoric acid; but there was no reason to suppose that the phosphorus was present in the organic principles in any other form than phosphoryl (PO).

The quantitative relations of the immediate principles and constituents of the brain are fully discussed from Dr. Thudichum's own standpoint, and synopses are given of analyses of the grey and white substance of the human brain. The mean sp. gravity of grey tissue is stated to be 1·032; of white, 1·041; and of the entire brain, 1·037;—numbers which differ slightly, or not at all, from those commonly received. Of white tissue he assumes there is present 55 per cent., and of grey 45 per cent. in the entire brain. A useful sketch of a systematic quantitative analysis of the brain is next laid down, and this is followed by a table showing the result of quantitative analyses of the human brain. From the latter we shall make a few selections. The quantities given, it should be said, claim to be minima only, while the losses registered are confessedly great.

HUMAN BRAIN.

	Grey tissue.	White tissue.
Water expelled at 95°	85·270	70·230
Neuroplastin	7·608	8·630
Kephalin, lecithin, myelin, &c.	2·730	11·497
Cerebrins and myelin	0·434	6·910
Inosit	0·193	0·215
Lactic acid	0·102	0·045
Phosphoric acid	0·017	..

"When the normal composition of the brain," as the author observes, "shall be known to the uttermost item, then pathology can begin its search for abnormal compounds or derangement of quantities. Thus the amyloid degeneration is specific to brain and nerve tissue, and can be considered hypothetically as a reduction of an ingredient of decomposed cerebrosides." The great diseases of the brain and spine, such as general paralysis, acute

and chronic mania, melancholia and others, the author believes, will all be shown to be connected with special chemical changes in neuroplasm. The knowledge of the composition and properties of this tissue and of its constituents will materially aid us in devising modes of radical treatment in cases in which at present only tentative symptomatic measures are taken. Indeed, there is great probability in his assumption that, by the aid of chemistry, many derangements of the brain and mind, at present obscure, will become accurately definable, and more amenable to precise treatment.

The opportunity may here be taken of referring to a paper, published by Dr. Thudichum a short time ago, on the chemical diseases of the brain and spinal cord. He here assumes, that from the chemical constitution of an organ we may deduce the pathological changes which must necessarily take place in it under certain conditions, particularly after the ingestion of drugs possessing special affinities for some of its constituents; and that treatment must be based upon a knowledge of these affinities. Locomotor ataxy, chronic alcoholism, lead and arsenic poisoning, are adduced as examples of these chemical diseases; and in connection with the same, some of his remarks on what he designates cerebrose diabetes, a kind of acute glycosuria, are highly interesting. But into these, or his criticisms as to amyloid degeneration, we cannot here enter, nor are we disposed to agree with his opinions as to the constitution of amyloid substance.

Dr. Thudichum thinks it highly probable that phosphorus, an ingredient present in the brain in such a small proportion, may yet be its most indispensable constituent. Phosphorised substances are found in every centre of vital activity, and seeing that they can combine, in the state in which they exist in the nervous system, with almost all the chemical reagents with which they are brought in contact, provided the latter are in sufficient relative excess, these combinations in turn readily breaking down again, it is highly probable that they are closely concerned with much of brain disease. Lead-poisoning, he observes, being regarded as nerve poisoning, the kephalin and myelin combining with this body, the object of treatment must be to break up these combinations by the administration of a body capable of removing the lead from its combinations. The combination with myelin is very stable, that with kephalin quite unstable. Both these bodies have acid characteristics, the myelin behaving as a dibasic acid; while in apomyelin and lecithin the alkaloidal character prevails, and accordingly they combine more readily with such reagents as cadmic or platinic chlorides. Now, in the case of chronic lead-poisoning, potassic iodide has long been recognised as an efficient curative agent, and it is further known that it acts more readily when large quantities of water are administered with it. When a solution of this salt is brought in contact with a solution of a lead salt, iodide of lead is at once formed and potassium set free; and this is what occurs when it is given in cases of lead-poisoning. The water administered now plays its part by deluging the vessels

with a solution of potash, which is not only a highly diffusible liquid, but also a stimulant of liquid secretion. The lead iodide is soluble in the potash solution, and as fresh quantities of potassic iodide are being constantly added, the lead combinations are more and more broken down, and thus the system is gradually freed from its presence.

Many kinds of headache, according to the author, are probably due to intracranially brewed chemical poisons, or to poisons carried to the brain by the blood from the body, having been fermented there, or, like alcohol, morphia or fousel oil, formed outside the body. "Many forms of insanity are unquestionably the external manifestations of the effects upon the brain substance of poisons fermented within the body. These poisons we shall, I have no doubt, be able to isolate after we know the normal chemistry to its uttermost detail. Then will come, in their turn, the crowning discoveries to which all our efforts must ultimately be directed, namely, the discovery of the antidotes to the poisons, and to the fermenting causes and processes which produce them."

Very much of the chemistry of the brain has, in our opinion, been omitted from the author's present treatise. He has neglected almost all reference to the work of anybody but himself. With much self-satisfaction, however, he thus excuses himself: "The literary discussions on subjects of brain chemistry which have taken place during the last few years, and in which I was obliged to take an active part, have had such an issue that I was happily enabled to exclude all controversy from the pages of my treatise." Accordingly, while we are disposed to regard Dr. Thudichum's treatise as a valuable contribution to the chemistry of the brain, we certainly cannot look upon it as embodying what is known as to its chemical constitution: and time alone will tell whether or not his contributions will be received as part of the great foundations upon which future researches may be safely superimposed.

T. Cranstoun Charles, M.D.

Mr. Sydney Hodges *on Retinal Images.*—I. "*After-Images.*" 'The Nineteenth Century,' October, 1883. II. "*On some Peculiarities connected with Retinal Images.*" 'Brain,' April, 1884.

The repetition by accurate observers of experiments previously and even repeatedly made by others is seldom altogether without value. When therefore Mr. Hodges has been led by an accidental occurrence to make a careful and able study of the after-images resulting from the fixation of very luminous objects, it might seem unnecessary to point out the large amount of work already done covering the same ground. Were it not indeed that Mr. Hodges

expressly urges the prosecution of investigations such as he has undertaken, as likely to be productive of novel and interesting results, we need hardly have referred to this subject in the pages of 'BRAIN.' But it is important to remember that such experiments are not unattended with danger. Two eminent physicists, Plateau and Fechner, had occasion to regret the ardour with which they prosecuted researches of this nature. The former, with the view of deterring others from running the same risk, which cost him so dearly, not only repeatedly gave warning of the danger, but took the trouble to make a very complete bibliography of this subject.[1] A reference to that bibliography will convince any one that the main facts with regard to the effects produced by experiments of the nature of those under consideration, as far as they give rise to sensational changes, are well established. There is so close an agreement in the chief results described by most observers, that no further confirmation is required. So far as any theory or explanation of the colour-sense is concerned, these are available, and must undoubtedly be taken into consideration. Almost all modern physiologists who have occupied themselves much with the subject of visual sensations, such as Helmholtz, Hering, Brücke, Exner, Fick, Aubert, and a host of others have given more or less attention to this subject. Some, indeed, have advanced it far beyond the stage to which Mr. Hodges has arrived in his researches, and in ways which to explain would hardly be suitable matter for the pages of a neurological journal, but which those interested in the theories of vision will find to throw much more light on their speculations. It is true that Helmholtz in his 'Physiological Optics' does not devote much space to Mr. Hodges' subject, but he will find in it at least one very definite statement which I quote from the French edition, § 23. He there remarks: 'Les objets colorés ne sont pas les seuls qui donnent des images accidentelles colorées, les objets blancs en fournissent aussi, qui présentent ordinairement des modifications de couleurs très variées. Nous donnons à ces variations le nom de phases colorées des images accidentelles. La succession de couleurs diffère avec la durée et l'intensité de la lumière première."

The phenomena seem to have been first correctly described by Porta in 1593, though observed by Aristotle and Themistius, the

[1] Plateau: "Bibliographie analytique des principaux phénomènes subjectifs de la vision depuis les temps anciens jusqu'à la fin du 18me siècle, suivie d'une bibliographie simple pour la partie écoulée du siècle actuel."—*Troisième Section.* "Images qui succèdent à la contemplation d'objets d'un grand éclat, ou même d'objets blancs bien éclairés." (In which the writings of twenty-six authors on this subject up to the present century are analysed, and fifty-two others of later date cited.) This section, as well as the complete bibliography, is prefaced by the following warning: "Les expériences qui font l'objet de cette troisième section sont dangereuses; c'est à la suite d'une expérience imprudente de ce genre, que s'est développé chez moi le germe de l'affection qui a fini par me priver complètement de la vue. Je ne saurais donc engager trop fortement les physiciens et les physiologistes à s'abstenir de semblables essais, qui ne présentent qu'une importance bien minime à côté des maux qu'ils peuvent entraîner. D'ailleurs, les faits observés jusqu'ici sur cette matière sont, je le pense, assez nombreux pour qu'on puisse se dispenser d'en recueillir de nouveaux."

first three-and-a-half centuries before, and the second three-and-a-half centuries after Christ. Malebranche in 1699 not only described the order of colours, but observed that the transition from one colour to another was gradual, so that for an appreciable time he was conscious of seeing intermediate lines. It seems not, however, to have been till towards the end of the last century that the fact was well known that the colour of the after-image is, as Mr. Hodges says, "produced not by the tint of the object we look at, but by the amount of light thrown on the retina."[1] About the same time, it was also observed that even pure spectral hues, if of sufficient intensity, gave rise to the sensation of white light. Various theories were almost invariably advanced by those who conducted such experiments. Descartes considered that the successive changes which the after-images underwent showed that the nature of the colour-sense consisted in a variety of different movements. Michælius shortly afterwards, in 1645, declared that the images only existed in the imagination, and that the successive changes were due to a struggle between the imagination and the judgment; Kircher again explains the phenomena as analogous to fluorescence. Most of the authors who wrote before the present century agreed more or less with Descartes. It is only lately, since the discovery of the visual purple by Boll, that so many have sought for an explanation in photochemical changes taking place under altered or more or less disturbed circumstances. The phenomena described by Mr. Hodges, when only one eye at a time is subjected to the stimulus of strong light, are the well-known phenomena of "Contrast of the fields of vision," excellent monographs on which have been written by Prévost and Panum.

GEORGE A. BERRY, M.B.

[1] See Plateau's references to Darwin and Venturi.

Abstracts of British and Foreign Journals.

Schultze on Secondary Degenerations in the Spinal Cord.—The author (*Archiv f. Psych.* Bd. xiv. p. 359) bases his paper on the following five cases:—(1) Compression of the cauda equina; (2) crushing of the lower half of the lumbar enlargement and the sciatic portion of the cauda equina; (3) atrophy and degeneration of the greater part of the lumbar enlargement, the result of fracture of the 1st lumbar vertebra; (4) breach of continuity in the cord at the level of the 9th dorsal vertebra; (5) perimeningeal tumour in the cervical region with atrophy of the middle third of the cervical enlargement from pressure.

First, as regards degeneration in the posterior columns. Schultze finds that immediately above the seat of injury the posterior columns degenerate in their entirety; further upwards the degeneration is confined to the inner portions of the columns, in an especial manner to Goll's columns. This rapid diminution in the extent of the degeneration is due to the fact that two kinds of fibres are represented in the posterior columns; a short set of fibres that spring from the posterior spinal roots and soon end, probably in the cells of the posterior cornua; and a long set that pass upwards to the clavæ of the medulla oblongata. Goll's columns are composed of fibres of this last set, and chiefly of those that spring from the posterior sensory roots of the nerves that supply the lower extremities, though perhaps also of those that spring from the posterior roots in the lower dorsal region. A degree of localisation is now possible within the limits of Goll's columns: for the innermost and hindermost portions of the columns are shown by Case 2 (in which the only symptoms were those of complete paralysis of the sciatic nerve) to be occupied by fibres that belong to the sciatic nerves. Pathology teaches us that Goll's columns have a much larger area in the cervical portion of the cord than Flechsig gives as a result of his embryological studies. Schultze remarks that the extent of the secondary degeneration is not always proportional to the extent and severity

of the symptoms; which is probably due to the fact, that the area of degeneration is generally determined not by microscopical but by macroscopical examination, and this may easily lead to considerable error.

Descending degeneration was observed in the posterior columns in Case 5. Schultze has seen it in other cases, and Westphal, Kahler and Pick, and others have described it. It extends only 2 or 3 ctm. downwards. Schultze suggests that it is caused by degeneration of those fibres of the posterior roots that bend downwards as they enter the posterior columns.

Secondary degenerations in the lateral columns are both ascending and descending. Of ascending degenerations the most important is degeneration of the cerebellar tract. This was found after lesion of the lower dorsal cord, not after lesion of the lumbar enlargement and cauda equina. The tract as marked out by degenerations reaches further forwards that Flechsig represents it. Schultze has not observed the punctiform degeneration in the central parts of the lateral columns that Singer has described in dogs. In one case he noticed a partial degeneration of the anterior half of the column, but is doubtful whether this was a true secondary degeneration.

The author has nothing new to say of descending degenerations of the pyramidal tracts. In Case 5 there was descending degeneration not only of the pyramidal tracts, but to a less degree of the whole lateral columns, with the exception of their innermost portions (the lateral limiting layers), and also degeneration of parts of the ground bundles of the anterior columns: the disease extending 8 or 10 ctm. downwards.

The paper concludes with some remarks on the pathological anatomy of tabes, which Schultze regards as primarily an atrophy of the nerve fibres.

Westphal on Spastic Spinal Paralysis.—Westphal (*Archiv f. Psych.* Bd. xv. p. 224) reports a case which presented the following symptoms:—Spastic paresis of the lower extremities, spontaneous twitchings of the muscles, heightened tendon-reflex, diminished sensibility to temperature, somewhat increased sensibility to pain; and the act of micturition was rather painful, and caused a powerful tonic convulsive movement throughout both lower limbs. Westphal remarks that the sudden forcible bending of a joint will often give us indication of an incipient spastic contracture, when slow, gradual passive movements tell us nothing. The case is

remarkable, in that the symptoms remained stationary for about four years, when paralysis rapidly developed in the right arm, brain symptoms showed themselves, and death occurred in four weeks. It cannot be considered a typical case of spastic paralysis, on account of the bladder troubles and sensory symptoms.

The post-mortem disclosed primary degeneration of the pyramidal and direct cerebellar tracts of the lateral columns of both sides; slight irregular degeneration of Goll's columns in the cervical and upper dorsal regions; and softening of the central white matter of the hemispheres, particularly in the left cerebral hemisphere. There was hypertrophy and dilatation of the bladder. This case is in accord with Westphal's previous experience, that the symptoms of spastic paralysis may be associated with disease of the lateral columns, conjoined with disease of the posterior columns, if the latter does not extend to the lumbar portion of the cord.

Westphal returns to the subject of uncomplicated primary sclerosis of the pyramidal tracts of the lateral columns, and controverts Flechsig's statement, that the so-called cases of this affection in general paralysis, are really cases of descending degeneration of cerebral origin. According to Westphal, it is in cases of general paralysis, and in these alone, that this peculiar form of sclerosis has yet been found.

Vierordt on Combined Disease of the Anterior Cornua and Lateral Columns of the Cord.—Vierordt (*Archiv f. Psych.* Bd. xiv. p. 391) reports a case of which the following were the chief symptoms:—There was a gradual appearance of weakness in the muscles of the extremities, with subsequent atrophy of the muscles; there was the reaction of degeneration; there was no trace of muscular contracture; the patellar reflex was retained; sensibility and the functions of bladder and rectum were normal, and there were no bulbar symptoms. The patient died of an intercurrent attack of bronchitis. There was advanced degeneration of the grey matter of the anterior cornua of the cord; degeneration evidently more recent of the pyramidal tracts of the lateral columns from the lumbar region to the decussation, but not beyond this; degeneration in a moderate degree of the anterior mixed zones of the lateral columns; and degeneration of the motor nerves and of the muscles. The direct cerebellar tract and the lateral limiting layer were normal. Disease of the anterior mixed zone of the lateral columns has been noticed by many authors in cases of combined disease of

the anterior cornua and lateral columns. It is also found in some cases of poliomyelitis anterior, and progressive muscular atrophy.

This case is an example of primary degeneration of the ganglion cells and nerve-processes of the anterior cornua, with secondary systematic degeneration of the pyramidal tracts. A remarkable feature is the absence of degeneration of the pyramidal tracts above the decussation. But notwithstanding the absence of bulbar symptoms, the case is closely allied to Leyden's amyotrophic bulbar paralysis. They are both characterised by degeneration of pyramidal tracts with flaccid atrophic paralysis, and so stand in contrast with Charcot's amyotrophic lateral sclerosis, in which there is degeneration of the pyramidal tracts, but rigid paralysis with contractures.

Hoffmann on Acute Ascending Paralysis.—Typical cases of acute ascending paralysis are characterised, according to Westphal and others, by an absence of morbid appearances in the cerebro-spinal nervous system; and the theory has been advanced that the disease is due to some toxic agency. On the other hand, cases have been described in which there was a well-marked affection of the cord and medulla oblongata; and this has suggested the view that the disease belongs to the group of acute myelitis. It is as a contribution to this discussion that Hoffmann reports his case, *Archiv f. Psych.* Bd. xv. p. 149. Clinically, it was a typical case of the disease. Microscopic examination disclosed inflammatory changes in the cord and medulla oblongata. There was inflammation of the pia in the cervical and dorsal regions, with patches of myelitis, especially in the lateral and anterior columns, and small hæmorrhagic extravasations; similar changes were observed in the medulla, in particular in the pyramids and corpora restiformia. This case then supports the myelitic view of ascending paralysis.

Strümpell, Vierordt, and Müller, on Multiple Degenerative Neuritis.—The case that Strümpell reports (*Archiv f. Psych.* Bd. xiv. p. 339) was in its early stage diagnosed as a chronic poliomyelitis. There was extensive and rapidly developed paralysis in all four extremities, with marked atrophy of the affected muscles, and the reaction of degeneration; the muscles of the trunk were paralysed, the respiratory muscles and the diaphragm last of all; the functions of bladder and rectum were normal; there was no decubitus. The only symptom that threw

doubt on the diagnosis was the slight sensory affection; there were in the early history of the case darting pains in the arms and legs, and during the latter stages analgesia, and at other times hyperæsthesia in the lower extremities. The presence of sensory symptoms will probably be an important point in the differential diagnosis of poliomyelitis and multiple neuritis. Other interesting symptoms were the persistently high pulse-rate, which was never under 120 per minute, and which may perhaps be attributed to an affection of the vagus, the œdematous swelling of the four extremities, and the commencing atrophy of the optic nerve.

The autopsy showed degenerative atrophy of the peripheral nerves and muscles. The anterior nerve-roots of the spinal cord were quite normal. The term degenerative *neuritis* is used, but there was no trace of even old inflammatory changes, and the disease is probably simply a nerve degeneration. The symptoms indicated that the motor nerves were implicated in a much larger degree than the sensory nerves; but the *post-mortem* does not throw any light on this point, which is deserving of special elucidation in future cases.

As regards the etiology of the disease, there is reason to suspect that it owes its origin to some infective source. Beri-beri, which is similar in many respects to the disease under consideration, has been shown to be a multiple neuritis, and it is endemic in many tropical and subtropical places. Tuberculosis is a frequent complication in cases of multiple neuritis. Strümpell thinks that many cases of acute ascending paralysis (Landry's disease) are really cases of multiple degenerative neuritis, and he points out the necessity of examining the peripheral nerves and muscles in all cases.

Vierordt (*Archiv f. Psych.* Bd. xiv. p. 678) reports a case of this disease in a patient suffering from phthisis, and comments on the frequent association of the two diseases. He also carefully describes the different sensory symptoms, and indicates their relative diagnostic importance.

Müller (*Archiv f. Psych.* Bd. xiv. p. 669) publishes another case, complicated with tuberculosis and mental symptoms.

Greiff on the Localisation of Hemichorea.—Two cases are reported (*Archiv f. Psych.* Bd. xiv. p. 598). The first was a case of left hemichorea following left hemiplegia, and the autopsy revealed lesions in the left cerebellar hemisphere, the right occipital lobe (implicating the occipito-temporal, lingual, and inferior

temporal convolutions) and the right optic thalamus. In the optic thalamus there were two small hæmorrhagic foci, one in its upper and inner portion, the other at its lower border where it is contiguous with the pes pedunculi, into which the focus could be traced. Greiff concludes that it was this last lesion that caused the hemichorea by the excitation it produced on the pyramidal fibres of the peduncle.

The second was the case of a man, aged 51, who presented in the early part of his illness the symptoms of senile dementia, but afterwards epileptiform attacks developed, so that the case may be referred to the group of progressive paralysis (general paralysis). After one of the convulsive attacks, in which the left side had as usual been most affected, left hemichorea appeared, and lasted four days, when it declined into a hemiathetosis, and passed away. Microscopic examination showed, in an extreme degree, changes in and around the vessels with pathological alterations of the nerve-cells. These changes were most marked in the right central convolutions and the paracentral lobule. There was also a small focus of incipient softening in the upper part of the right pons adjacent to the pyramidal strands, with descending degeneration of these strands. It is impossible to decide which of these lesions determined the hemichorea, but in either case it was probably by irritation of the pyramidal fibres.

Unverricht on Epilepsy.—Unverricht (*Archiv f. Psych.* Bd. xiv. p. 175) summarises the results of an experimental and clinical investigation into epilepsy under twenty-five heads. We can only notice a few of his conclusions. He distinguishes two varieties of the status epilepticus, experimentally produced: the oscillating variety, in which the spasm travels from one side of the body to the other, and then returns by the way it came, and so swings pendulum-like from side to side until the convulsion becomes general; and the variety in which there is a series of spasms commencing in one place and taking the same course, and following each other with ever-increasing rapidity, until they become general. The body temperature may be lowered to 34·8° C. without affecting the irritability of the cerebral cortex or the appearance of the status epilepticus. Anæmia and asphyxia only arrest convulsions when they are pushed to a degree that endangers life. Injections of chloral into the veins, or the inhalation of ether, readily arrests them. Extirpation of the various motor centres frees the corresponding muscles from participation in the

convulsive seizures, though associated movements may still occur. Extirpation of the motor areas during the status epilepticus arrests the convulsions in the muscles supplied by these areas. Division of the corpus callosum does not prevent the spreading of the convulsion from one side to the other. Hæmorrhage into the ventricles arrests the convulsions on the opposite side of the body.

Zacher on General Paralysis.—Zacher (*Archiv f. Psych.* Bd. xiv. p. 463) continues his paper on some peculiar forms of general paralysis (for abstract of first part of which, see 'Brain,' Vol. V. p. 566).

(1) *On the Tendon and Skin Reflexes during and after Paralytic Attacks.*—The author made observations on general paralytics with healthy spinal cords, with fascicular sclerosis of the lateral columns, and with combined disease of the posterior and lateral columns, respectively; and he comes to the conclusion that the tendon-reflexes during and after paralytic attacks are increased on the side which presents convulsive symptoms, even though a degree of paresis coexists, but are diminished or even abolished on the side which presents simply flaccid paralysis. If the tendon-reflexes were increased before the attack, they will either be still further increased or diminished, according to the above rule. If they were absent before the attack, they will not be re-established. A similar relation exists between the superficial reflexes and the condition of sensibility. Zacher accepts Schwarz's theory as to the correspondence between brain and spinal centres (see 'Brain,' Vol. VI. p. 417).

(2) *On certain Motor Phenomena during and after Paralytic Attacks.*—Muscular tension, rigidity, and contracture are often observed, and they probably express merely a different degree of the motor irritability that gives rise to convulsive phenomena. They are not to be looked on as evidence of spinal lesion, since they may be present where the cord is sound; they are probably produced by excitation of the cerebral cortex. Another motor phenomenon observed during these paralytic attacks is that of automatic, purposed co-ordinated movements, that have all the appearance of being directed by the will. Similar movements are familiar to us in some epileptic conditions and have been described by Fürstner in pachymeningitis. The presence of paræsthesia and ill-defined sense-impressions may account for the movements, or on the other hand they may simply be the result of a primary excitation of a motor area. Another of the motor phenomena alluded to by

Zacher is the conjugate deviation of the head and eyes, which follows the laws laid down by Prévost and Landouzy. And lastly, he draws attention to the occurrence of disturbances of the muscular sense, evidenced by the patient's want of knowledge as to the position of his limbs.

(3) *On Visual Disturbances during and after Paralytic Attacks.*—These may be divided into two classes. First, cases of pure mental blindness (*Seelenblindheit*). In the few cases Zacher has observed, the blindness was bilateral, though the motor symptoms were only on the right side. Secondly, cases of bilateral visual affection, which are probably examples of true hemianopsia, and are due to lesion of the posterior cerebral lobes. To these should be added perhaps a third class, including cases presenting a combination of these various visual disturbances, such as Fürstner and others have described. The existence of unilateral amaurosis of cerebral origin must still be regarded as unproved.

(4) *On some peculiar Vaso-Motor Symptoms.*—If a blunt object, such as a key, was drawn across the skin, a pale streak was seen, which soon became intensely red, and close to it appeared little square raised patches, which soon coalesced and formed a strip, slightly raised above the general surface. This is probably an exaggerated example of the phenomenon to which Trousseau drew attention, and which is exquisitely seen in some epileptic conditions.

Siemens on Refusal of Food in the Insane.—The author (*Archiv f. Psych.* Bd. xiv. p. 569, Bd. xv. p. 15) is of opinion that the refusal of food is by no means the serious symptom it is generally considered to be, and he makes the somewhat startling statement that abstention from food, if purely mental in origin, is attended with no danger, provided it is not prolonged over fourteen days, when no water is taken, or over fifty days when water is taken, and provided, too, that the loss of weight does not exceed 40 per cent. of the total weight. He is opposed to forcible feeding, except in cases of prolonged loss of consciousness and paralysis, on account of its mechanical dangers (injury to the fauces, pneumonia from food in air-passages), and also because it is unphysiological, taking no note of the digestive and assimilative capacities of the individual. He trusts chiefly to time, and tact, and the presentation of the kind of food the patient likes best. There is a class of cases, generally old hypochondriacal melancholic persons, which no sort of treatment, with the stomach-pump or without it, is able to save.

Langreuter on Paraldehyde and Acetal in Mental Diseases.—The author (*Archiv f. Psych.* Bd. xv. p. 1) recommends paraldehyde as a hypnotic in mental diseases. It is of most service in cases of epileptic excitement with feelings of terror, &c., and in the similar conditions of disturbed consciousness that occur in general paralysis. The superiority of the drug over chloral lies in its more rapid action and greater safety. The dose is from five to six grammes, and a sleep is produced in from five to ten minutes, that lasts about two hours, and often, especially at night, seven or eight hours. Acetal is similar in its action to paraldehyde, but it is not so reliable, and its smell and taste are much more disagreeable.

W. J. Dodds, M.D., D.Sc.

Wilks on Hemianæsthesia.—In vol. xxxvi. of *Guy's Hospital Reports*, Dr. Wilks makes a valuable contribution to the literature of this subject. He shows that, as is generally admitted, such cases do not admit of explanation by the hypothesis of a gross lesion; and gives reasons, moreover, why such lesion as there is cannot be in the region of the internal capsule. He emphasises the constant association of the phenomena of hemianæsthesia, hemianalgesia, &c., with the moral characteristics of hysteria; and counts this as one among other reasons for attributing what lesion there is to the cortex. He considers that the central circumstance in the malady is the arrest of the function of a large area, or the whole of the cortex of one hemisphere. Such cessation of action is common enough as affecting the two hemispheres simultaneously; as is evidenced by the occurrence of sleep, of stupor after blows, and other conditions. If we suppose such cessation to occur over part of the cortex instead of over the whole, it will account in great measure for the temporary one-sided losses and for the other manifestations attending them.

Gairdner on Lethargic Stupor or Trance.—An unusually well-characterised instance of this affection is related by Professor Gairdner (*Lancet*, Dec. 22, 1883, and Jan. 5 and 12, 1884). The patient remained in a completely helpless and apparently unconscious state from Aug. 1 to Nov. 18, 1883. During the whole of this time she did not once execute any movement save a slight movement of the eyelids. No response whatever was made to stimuli of the utmost justifiable intensity; and but for attentive artificial feeding the patient must have perished of starvation.

Such cases are probably closely allied to cases of hemianæsthesia. They may be looked upon as virtually cases of *double* hemianæsthesia, hemianalgesia and hemiplegia; and would, on Dr. Wilks's hypothesis, be looked on as due to loss of function of the entire cerebral cortex of both hemispheres.

CH. MERCIER, M.B.

Parant on Paralysis Agitans as a cause of Insanity. (*Annales Médico-Psychologiques*, July, 1883).—Dr. Parant relates the details of an interesting case of paralysis agitans, in which various manifestations of insanity appeared, and gives a résumé of what has been said by the chief French writers on this subject, by whom but little notice is taken of the mental side of the disorder in question; those who mention it at all, merely drawing attention to the dementia or general weakening of the mental faculties commonly supervening in the later stages. Professor Ball, however, has frequently seen mental disturbances in these patients, not only irritability and general mental weakness, but also true insanity with hallucinations and delusions, the insanity, in his opinion, generally assuming the form of melancholia, with delusions of persecution. Professor Ball and Dr. Parant both notice that when the tremor becomes less marked, the mental troubles also tend to disappear.

Dr. Parant compares paralysis agitans with miliary sclerosis and locomotor ataxy. He says, quoting Charcot, that in the cerebro-spinal form of miliary sclerosis, we generally, towards the end of the disease, find that the mental faculties become weakened, but that in some cases the classical forms of insanity present themselves. The sufferers from locomotor ataxy, also, occasionally become insane, the characteristic form here being melancholia with delusions of persecution and various hallucinations depending upon lesions of the optic, auditory, or other sensory nerves. The insanity in locomotor ataxy and miliary sclerosis is due to the extension of the morbid process from the cord to the brain; and Dr. Parant is of opinion that this is also the case in paralysis agitans. The writer thus formulates his conclusions:—

(1) That true insanity is occasionally met with in patients suffering from paralysis agitans; that it may present itself in various forms, but that mental depression is the predominant feature.

(2) That this insanity is probably due to the extension of the lesions of the cord to the brain, or to the formation in the brain of

isolated patches of disease springing from the same cause as the chief disorder in the cord.

Camuset on the prevalence of forms of General Paralysis in which Depression is the predominant feature. (*Ibid.*, May 1883.)—After examining a great number of general paralytics the author has come to the conclusion, that depression is more frequently met with in this disease than exaltation. He says that general paralysis presents itself under three chief forms: (1) Simple paralytic dementia; (2) paralytic dementia with exaltation; (3) paralytic dementia with depression; and on taking a general view of the cases of this disorder under his observation, he is struck by the fact that the patients are sad, melancholy and depressed; excitement, self-satisfaction and delusions of a grandiose character, being comparatively rare. He gives the result of his examination of 373 general paralytics in a tabular form, showing that cases characterized by depression are twice as frequent as those in which exaltation predominates.

R. Atkinson, F.R.C.S.

Bernhardt on Head-tetanus. (*Zeitschr. für Klin. Med.* Band 7, Heft 4.)—Patient, man of 32, had undergone, Oct. 17th, 1883, an operation for removal of a dermoid tumour on the left supra-orbitalis ridge. The wound healed quickly. On Oct. 22nd he had difficulty in opening mouth, and at the same time left-sided facial paralysis occurred, followed by pain in back and difficulty in swallowing. Tightness on the chest. Facial expression very peculiar, conditioned partly by the decided left-sided facial paralysis. Forehead smooth on left, and left eye open, nasolabial fold abolished, left corner of mouth drawn down. The movements of expression (*mimischen*), carried out on right side of face, did no take place on the left. In spite of left paralysis, expression of face strained and forced. Grinding of teeth loudly heard when he tried to open the mouth, due to convulsive contraction of masseters, which were tense and hard, especially on the left. In trying to swallow, convulsive reflex spasm was produced. Jaws firmly clenched, and muscles of swallowing spasmodically convulsive. The scar from the recent operation was one centim. distance from the left external corner of the eye in a slanting direction upwards, and was not particularly tender. Mind clear, behaviour anxious, pale face, speech normal, movements of eyes free in all directions, no squint, no double sight, pupils equal and moderate size.

Electrical excitability well marked with both currents in direct and indirect excitation of root and branches of the facial nerve on the paralyzed left half of face; with the same currents as those applied to healthy side, instantaneous contractions followed, except left frontal muscle which with direct excitation of the constant current, reacted to a weaker current than on the right side, but with distinctly slower contractions,

viz. Ka Sz = $1\frac{1}{2}°$ of the galvanometer,
Ka Oz = 3°, and A Sz by a current, not appreciated by the needle, but in striking contrast to the right side, where Ka Sz did not occur at $2\frac{1}{2}°$, A Sz only by much stronger currents, and Ka Oz not at all.

In region of chin and lower lip there is slight loss of sensibility to touch. No rigidity or paralysis of extremities. Tetanus, or rather head-tetanus, diagnosed shortly before death, when involuntary convulsions occurred, and pulse soft, compressible, temperature raised. State of patient continued the same till death in hospital on 2nd November following.

Post-mortem.—Pia of brain had a cloudy appearance on convexity of brain, otherwise macroscopically nothing to be seen abnormal. Pons and medulla not yet examined. Pharynx and œsophagus intact. Some branches of N. fac. sinister, also of right facial nerves examined microscopically and found intact.

The region under scar on left upper border of left eye cavity, extravasated with blood.

The course of the disease here described agrees exactly with the description given thirteen years ago by E. Rose in his treatise on tetanus of a peculiar type, which he has called "Head tetanus, or tetanus hydrophobicus." These cases of tetanus according to Rose occur after wounds in the region of the 12 cranial nerves; a marked characteristic of this is the intense spasm of the pharynx, producing a strong resemblance to hydrophobia. Rose says "it is probable that these cases of head tetanus, tetanus hydrophobicus, as they occur particularly after damage in the region of the central nerves, allying themselves particularly with spasms of central nerves, and of the pharyngeal muscles, have misled eminent practitioners to identify tetanus with hydrophobia. No one, who has seen such cases of head-tetanus, would find a resemblance between severe hydrophobia and acute tetanus."

Besides these spasms of the pharyngeal muscles, the presence of the facial paralysis appearing on the same side as the original

wound, especially stamped the disease as tetanus. Perhaps the affected nerve is swollen in the direction from the wound to the nerve centres: the paralysis would then be explained by the pressure in the unyielding fallopian canal. Rose has himself observed two cases of this kind, one ending in death, on which no light was thrown by autopsy, the other recovering.

In the above case the relation of the frontalis muscle to direct galvanic excitation was peculiar. The phenomena of degeneration-reaction was here shown: but it remains doubtful whether this reaction pointing to a change of the one muscle was dependent on a deeper affection of the facial root, or upon a local lesion of this particular frontal branch only occasioned perhaps by the operation. Dr. Gluck, the operator, stated that no section of nerve had taken place in removing the tumour; an exact electrical examination of patients in the second and third week of the disease would alone determine whether the facial paralysis in head tetanus is always slight (in the electro-therapeutical sense) or whether so-called intermediary forms occur.

Professor Bernhardt next refers to the "tension" of the features in tetanic cases, which has been remarked on by Romberg, Hesse and others. According to Koenig the patient's face expresses grief, or fatigue accompanied by a grinning appearance, according to the preponderance of the muscles of the upper or lower half of the face. The muscles of the middle portion of the face are more or less stretched by the powerfully contracting upper or lower muscles. Although Koenig does not refer to paralysis of the muscles of the face, he distinctly urges having observed these things in several chronic cases, the starting-point of which was a wound in the head or face. Rose further cites other cases of tetanus after head wounds, where besides spasmodic contraction of the pharynx, contractions of the muscles of the face with convulsive movements of the tongue occurred (case of Guastalla), and earlier still in 1853, Pflüger refers in his work, "On the functions of the spinal cord in vertebrata, &c.," to a boy with trismus and spasms of pharynx and face after a contused and incised wound of the inner part of right eyebrow, the trismus extending to the muscles of whole body, especially the right side, recovery ensued; also to a case of Larrey's, where tetanus came on 9 days after a wound to the right frontal region in a soldier, beginning in the right eyelid; and was relieved by cutting through the right orbicularis oculi muscle; another patient of Larrey's had tetanus hydrophobicus, after a wound of the nose, with persistent spasm of the face muscles.

Bernhardt adduces cases showing that contracture and paralysis can occur at the same time in the muscles of the affected side of the face, due to reflex spastic conditions although voluntary impulses are in abeyance. He considers that the motor branches of the trigeminus are strongly affected in every case of head tetanus, and follow Pflüger's laws, viz.: that in reflexes occurring on both sides of the body, the muscles most affected are found on the same side as the excited centripetal fibres. Sometimes tactile sensibility on the wounded side of the face is affected, analogous to the motor paralysis of the face; the nerves and muscles presiding over swallowing are particularly affected, hence the term of tetanus hydrophobicus of Rose.

Of 8 cases, 3 recovered completely after long duration.

Of 4 of the fatal cases, characteristic pathological changes in the central nervous organs were negative.

Bernhardt thinks that possibly changes in the facial muscles may be found.

Finally, he remarks that the term tetanus hydrophobicus or head tetanus does not sufficiently represent the disease which, in addition to the severe spasm of the pharynx, has, as its characteristic, the paralysis of the facial muscles on the same side as the wound, but he cannot suggest a better name.

He refers to two cases, one by Nankivell in the *Lancet* for July 14th, 1883, after a wound of the bridge of the nose, ending in death; the other by Bond in the *Brit. Med. Journal* for Nov. 10th, 1883, which recovered.

Bernhardt concludes that (1) all the cases do not necessarily end in death; (2) the cases of complete facial paralysis, which recovered, did not belong to the most severe forms; (3) the term tetanus hydrophobicus cannot be applied to all the cases, as spasm of the pharynx is not always present; (4) complete facial paralysis on the same side as the wound is an extremely significant characteristic of all these cases.

Franz Riegel on Spasm of the Muscles of Respiration (*Zeitschr. für Klin. Med.*).—A peculiar case of spasm of the respiration lasting a year. The patient had undergone varied treatment, until brought into the Klinik (Giessen), when a complete cure was effected in a few days.

Of the three types of spasm, viz. (1) affecting the inspiratory, (2) the expiratory muscles, and (3) complicated respiratory spasms, in this case the expiratory muscles were affected.

K. Q., 14 years old, of nervous temperament, fell into the water in winter time. Afterwards he complained of pressure and pain at the epigastrium—in 14 days' time difficulty in respiration came on.

He complained of want of breath, difficulty in breathing, i.e. expiration. Internal organs and nervous system normal.

He had long pauses of breathing, of 20–30 seconds, followed by long inspiration and then long expiration, followed immediately by successive spasmodic expirations without intervening inspiration.

No diminution of thorax, but epigastrium violently drawn in and abdominal muscles severely contracted during the spasm.

Pressure on phrenic nerves and vagus produced no change in this breathing-rhythm.

Diagram of breathing taken by a canula inserted into the nostril.

First week after admittance to hospital, no treatment purposely given. No organic disturbance of any kind being found, and the boy perfectly healthy—a hysterical affection was suspected.

Psychical treatment resolved on. The boy was told the spasms could and must be suppressed—was made to breathe methodically and slowly—great improvement produced. To complete the cure he was told the application of the electric current was necessary. One application of faradic current sufficed to cure him. In 14 days' time no recurrence of spasms. This prompt success of a purely psychical treatment was a proof of the hysterical nature of the affection. Riegel further quotes Leyden's opinion, that spasm of respiratory organs is common in boys at the time when the voice changes.

Bernhardt on Partial loss of Sensibility (*Berlin Klin. Wochenschr.* 1884, No. 4).—The patient, a man aged 19, had gradual loss of feeling for four years, to pin-pricks, electric brush, heat and cold, in the right upper limb, the right shoulder, the right half of neck and head, bounded in front by the median line, behind by the middle of scapula and nape of the neck, above by the median line of head, and below by the fourth rib; the right side of the face (especially the forehead, nose, cheek, and lip) was very much less affected, this area being sharply defined by the edge of the lower jaw; the right ear, however, was as much affected as the right side of the neck. There was no motor paralysis, but some loss of power in the arms, and in the right more than the left.

No sign of hysteria. Legs often become numb when they are crossed. Organs of sense intact, on both sides. Power of both arms not quite so good as formerly.

There was a swelling on the posterior surface of the right elbow, he cannot flex the right elbow-joint properly, and crepitus is felt when the forearm is pronated and supinated; this is due to a fracture of the upper end of right ulna which occurred suddenly with moderate exertion, and subsequently the upper end of radius was dislocated from the ulna.

In the affected area tactile impressions are felt immediately, but not quite so clearly as in the left arm. Pressure sensibility and muscular sense normal; no ataxia. In the right upper arm pain for the strongest faradic current is abolished, but he feels the tension and drawing produced by the current. Pin-pricks felt as touches, but no retardation of impressions of touch. Mucous membrane of right conjunctiva, nose, and mouth, normal. Does not feel the pain of an electric spark on the affected area, and not appreciate boiling water there; but appreciates heat a little on the right face.

No atrophic changes except the fractures and dislocation which point to trophic changes in the bones.

Excluding other lesions, Bernhardt thinks that the lesion is in the right posterior roots of the cervical nerves from I. to VIII., or rather of all the posterior grey matter of the whole cervical cord, where the posterior roots enter.

Following Schiff's theory, that the tracts for transmission of pain are in the grey matter, and those for touch in the white matter of posterior part of cord, he thinks the disease involves also the great and small occipital, and the great auricular nerves, which come off from the cervical cord and supply parts of the ear and face, and also that the right ascending trigeminal root is affected.

He refers to similar cases of Kahler, Fürstner and Zacher, and Schüppel. If the disease should involve the medulla oblongata, as in the above cases and that of Westphal, phenomena of bulbar symptoms would occur, and disturbances of sensibility in the part supplied by the trigeminus. Bernhardt thinks, it is not always possible to diagnose during life, a gradual progressive lesion of the grey central substance of the cord from a syringo-myelitis; he believes that it is possible in some. He considers it very difficult to make a differential diagnosis, when the muscles are affected and not the skin, the lesion being confined to the motor parts of the cord. Syringomyelia has been found on post-mortem without any symptoms during life, and, similar to slow growing tumours of the brain, causing no symptoms.

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